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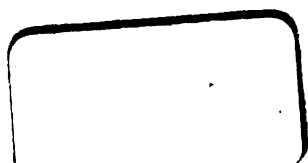
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SUPPLEMENT TO]

[APRIL 23, 1902.

THE
CLINICAL JOURNAL

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL
REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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IN TWO VOLUMES ANNUALLY.

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TENTH YEAR.

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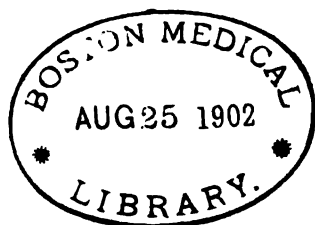
L. ELIOT CREASY, M.R.C.S.ENG., L.R.C.P.LOND.



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ON SOME CASES ILLUSTRATING THE SURGERY OF THE UTERUS.

By J. BLAND-SUTTON.

In this communication I have brought together a series of cases, some of which illustrate facts in pathology, others deal with points in operative surgery, but all have an instructive bearing on the diagnosis of uterine tumours, more especially in relation to cancer of the body of the uterus and fibroids. The cases are in many instances illustrated with drawings, and each picture is made as far as possible to tell its own tale, whilst the text serves in the main as a sort of running commentary. The bare narration of a case is rarely read, and the editor of one at least of our weekly medical journals shows his contempt for the excessive elaboration of clinical details by printing them in nonpareil instead of brevier.

CANCER OF THE BODY OF THE UTERUS.

It is a remarkable fact that cancer of the neck of the uterus is almost exclusively confined to women who have been pregnant (the exceptions in my observations are under 1 per cent.), and that repeated pregnancies appear to increase the liability of the cervix uteri to this disease. This accords with what is known of the natural history of cancer in other glandular organs, namely, *cancer is more liable to attack those that are injured in preference to those which are healthy.* In the uterus, however, we apparently meet with an extraordinary contradiction, for cancer of the endometrium is regarded as being more frequently associated with sterility. This has led me during the last four years to give some attention to the matter, and especially as it is now quite clear that the endometrium of the body of the uterus is liable to two varieties of cancer which differ in their clinical characters, minute structure, and what is much more important, in the results which follow upon operative treatment.

Cancer in the strictest pathologic sense is a disease of epithelium, and its minute structure is modified by the character of the epithelium in which it arises. This is well illustrated in the case

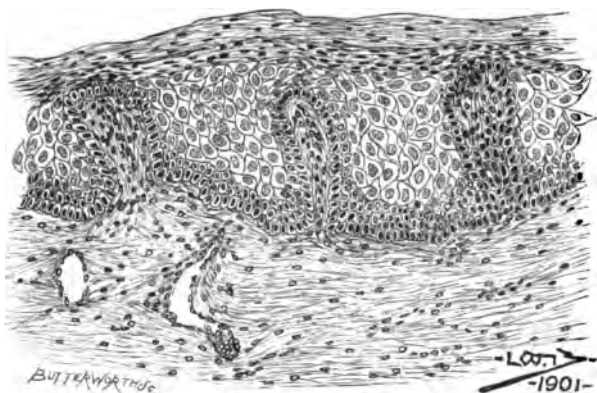


FIG. 1.—The microscopic characters of the epithelium covering the vaginal aspect of the neck of the uterus.

of the uterus. Cancer arising on the vaginal aspect of the cervix is of the squamous-celled kind (Fig.

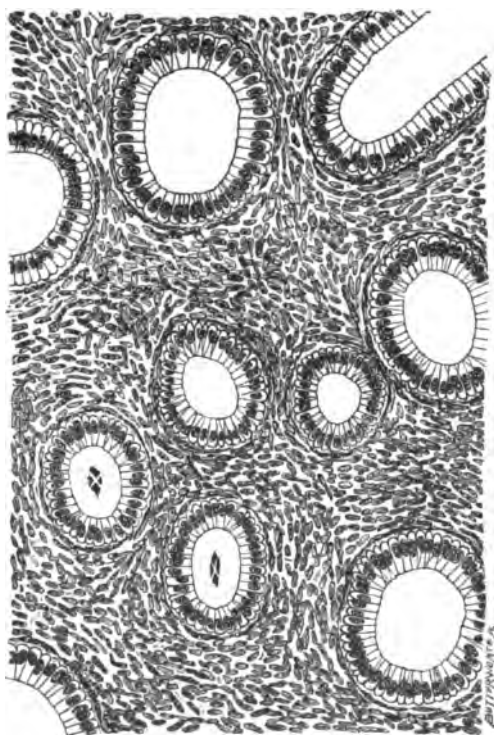


FIG. 2.—The tubular glands of the uterus in transverse section. (From the writer's book on 'Tumours'.)

1), whereas when it arises in the epithelium of the cervical canal it is of the columnar-celled species.

A comparison of the glands in the cervical endometrium and those of the endometrium proper is of some interest in relation to the cases to be considered later. The simple tubular uterine glands with their well-marked columnar epithelium furnish very striking appearances in sections of the endometrium when examined with the microscope (Fig. 2), whereas the glands in the cervical canal are racemose, and though lined with columnar epithelium, yet the cells are narrower than in the

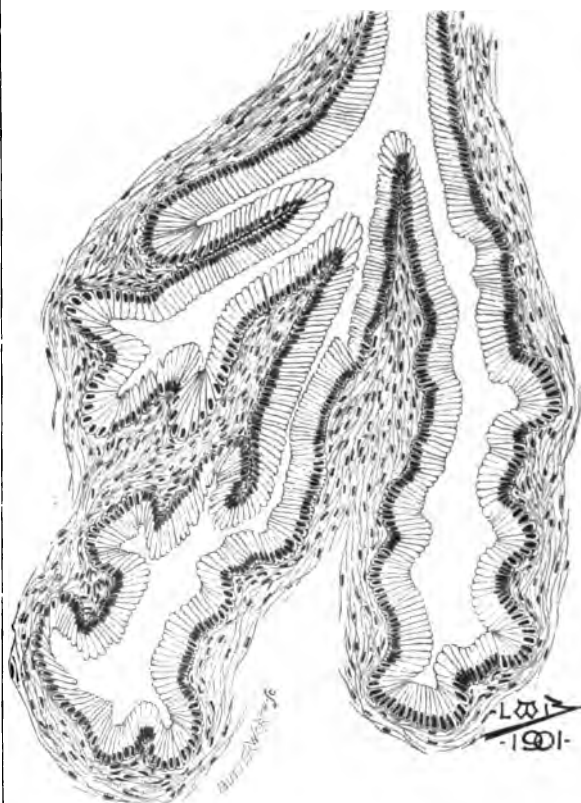


FIG. 3.—The microscopic characters of a gland from the cervical endometrium.

endometrium proper. It will easily be seen on reference to Fig. 3 that a section traversing a number of such glands would on examination furnish a more complicated picture than is presented in Fig. 2.

A large amount of energy has been devoted to the microscopic examination of cancer of the neck of the uterus, with the hope of determining the relative frequency of squamous-celled and of the columnar-celled species. So far as my own efforts are concerned they were directed with the object

of deciding if possible which species gave the best results to operation, but after a long and laborious investigation I came to the conclusion that it was hazardous to attempt a prediction simply on the cell features of the cancer.

Another important fact to realise is this :—Although in writings and bedside teaching we make a very sharp distinction between cancer arising in the cervix and cancer originating in the body of the uterus, many cases come under observation where it would be difficult to state with any certainty whether the cancer arose in the lower part of the body of the uterus or in the upper segment

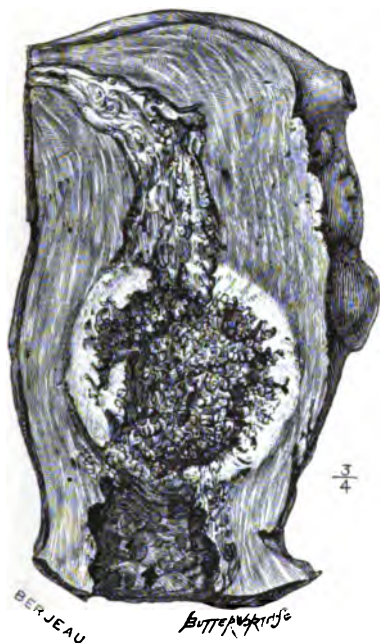


FIG. 4.—A cancerous uterus in coronal section. It was difficult to decide whether the cancer began in the upper part of the cervix or the lower part of the body of the uterus. A process of the growth is creeping into the right Fallopian tube.

of the cervix. Such a case is depicted in Fig. 4, and the uncertainty was not dispelled even on a most careful microscopic examination, for the histologic features of the cells and their grouping would do for either. An interesting fact in the case is the determined way in which the cancer has invaded the endometrium and crept along the cornu into the Fallopian tube. Although the uterus was completely removed the cancer returned about nine months after the operation and quickly destroyed the patient's life. She had been married three years, but was sterile.

In contrast to this the following case may be briefly mentioned. A spinster æt. 50 was placed under my care in 1898 for profuse and oft-recurring metrorrhagia attributed to a fibroid. On careful examination an interstitial fibroid the size of a fist could be made out near the right cornu of the uterus, but a careful consideration of the circumstance induced me to believe that the bleeding did not depend on the fibroid but rather on cancer of the endometrium, and on this view I recommended and performed abdominal hysterectomy. The uterine cavity was the seat of well-

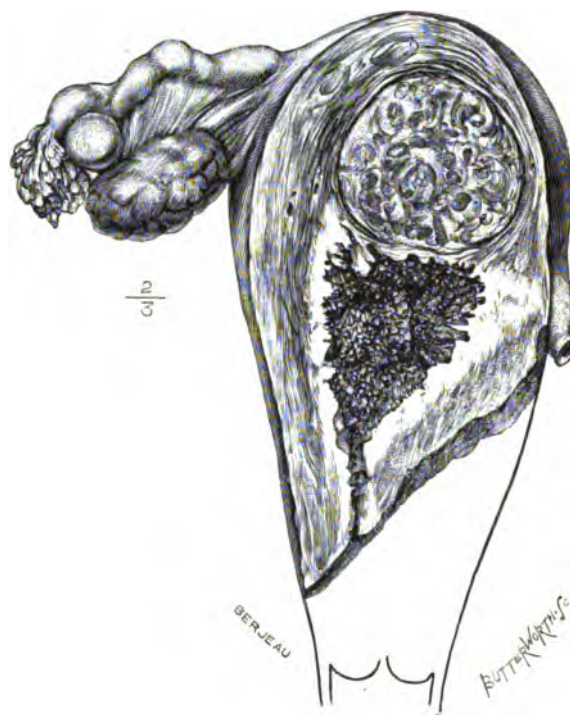


FIG. 5.—Cancer of the body of the uterus associated with a fibroid: successfully removed by abdominal hysterectomy from a spinster æt. 54 (Case 10 on page 5).

marked cancer which under the microscope showed no features by which it could be distinguished from the case last mentioned, but this patient is alive and in excellent health (Case 2 on page 5) three years after the operation.

In striking contrast to all these is the uterus represented in Fig. 6. The patient from whom this was removed, when she came into the hospital was thought to have a fibroid. She was bleeding profusely and profoundly anæmic; her uterus reached to a point midway between the symphysis

pubis and the umbilicus. It felt hard just like a fibroid. The patient was watched for a time and kept resting in bed, but even in such conditions she continued to bleed. I determined to remove the

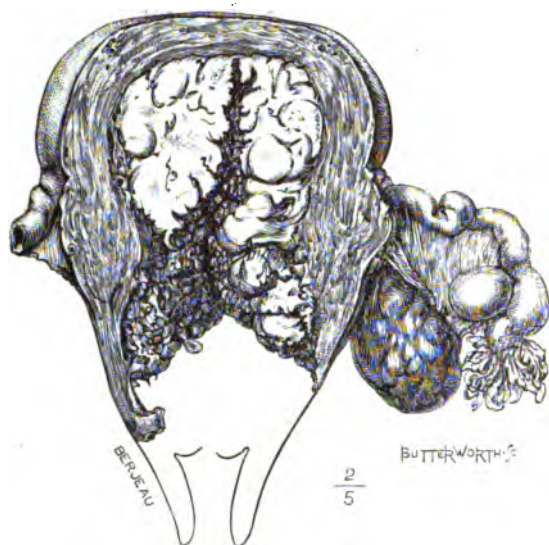


FIG. 6.—A cancerous uterus in section.

uterus by the abdominal route, and succeeded without any difficulty. On dividing the uterus our astonishment was very great to find the walls of the organ infiltrated with a soft brain-like growth and projecting into the cavity of the uterus in the form of tongue-like and polypoid processes. The general relation of the growth to the uterine structures is something quite different from the common forms of cancer of the endometrium, and its effects in enlarging the organ are shown when the scale on which the drawing was made is compared with that of Figs. 4 and 5.

In its microscopic features this growth is as widely different as in its gross characters from the vulgar forms of uterine cancer. A glance at the microscopic aspect of the common kind of cancer shown in Fig. 8 reveals it as sections of solid cell-columns, whereas in the uncommon kind the tubular character of the glands is markedly preserved (Fig. 7), and it is this striking structural feature which has led more than one recorder to call it "malignant adenoma," or by the still more misleading term "adeno-carcinoma." In order to appreciate its peculiar structure it is worth while to compare Fig. 7 with the drawing of the normal glands in Fig. 2 and of the common form of cancer

in Fig. 8; it will be at once remarked that in all the cut sections of the glands the central lumina are preserved, and the essential feature consists of an excessive multiplication of the glands as well as a marked increase in the epithelial elements; and these, as is so commonly the case with malignant epithelial growth, are very badly formed. In order to distinguish this species from the common species of uterine cancer I venture to suggest that it should be called tubular cancer, as expressing its most striking structural peculiarity.

When I first began to seriously study the diseases



FIG. 7.—Microscopic characters of the cancer in Fig. 6; the uncommon kind, or "tubular" cancer.

of the endometrium nothing could be more unsatisfactory than the fog of names which obscured the subject. I feel certain that the term villous endometritis covered many examples of carcinoma,

and especially the tubular species on account of the peculiar and fairly firm finger-like processes which are associated with it.

The importance of realising the differences in structure is the fact that this tubular species gives by far the best results to operative treatment.

Although cancer of the body of the uterus is happily far less frequent than in the cervical endometrium, it is nevertheless quite common enough, and I have appended a list of cases which have come under my observation within the last three years, in which the chief clinical features are set forth.

My interest in the question was at first directed to the circumstance that it is supposed to arise chiefly in aged spinsters or in those who have lived in sterile wedlock, but the subjoined table has

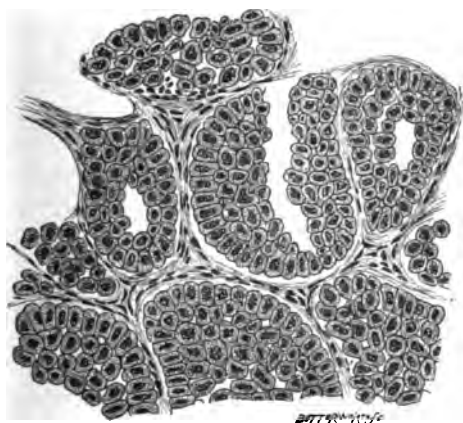


FIG. 8.—Microscopic characters of the common kind of cancer of the body of the uterus. (From the writer's work on 'Diseases of Women'.)

tended to show that this is not so exclusively a feature as we formerly believed.

CASE 1.—G—, æt. 56. Single and sterile. Vaginal hysterectomy, September, 1898, was followed by a quick recurrence and death, February, 1899.

CASE 2.—C—, æt. 50. Single and sterile. Abdominal pan-hysterectomy, October, 1898. The cancer was complicated by an interstitial fibroid as big as a fist. Patient in good health, October, 1901.

CASE 3.—O—, æt. 42. Single and sterile. Abdominal pan-hysterectomy, January, 1899. Patient in excellent health, October, 1901.

CASE 4.—S—, æt. 36. Married, but sterile.

Vaginal hysterectomy, November, 1899. Recurrence and death within a year. This is the case referred to in Fig. 4 as questionable whether it should be regarded as cancer of the upper part of the cervix.

CASE 5.—L—, æt. 45. Widow; sterile. Abdominal pan-hysterectomy, November, 1899. Reported in good health, June, 1901.

CASE 6.—D—, æt. 60. Single and sterile. Abdominal pan-hysterectomy, April, 1900. Uterus contained several fibroids the size of chestnuts; one was of the submucous variety, and embedded in the cancer. Patient in good health a year later.

CASE 7.—C—, æt. 54. Married; one child. Abdominal hysterectomy, June, 1900. The uterus contained a large fibroid, and the organ itself was so stuffed with cancer that the peritoneal surface presented numerous soft bud-like processes where the cancerous growth had eroded through the walls. No dissemination, but the iliac glands on both sides were infected. Patient died five months after the operation.

CASE 8.—H—, æt. 53. Married; six children. Abdominal hysterectomy, September, 1900. Patient in excellent health, July, 1901, and actively following her work as an obstetric nurse.

CASE 9.—G—, æt. 41. Married; one child. Abdominal hysterectomy, March, 1901. The uterus from this case is represented in Fig. 6.

CASE 10.—S—, æt. 54. Single and sterile. Abdominal hysterectomy, August, 1901. The uterus with the fibroid *in situ* is represented in Fig. 5.

CASE 11.—A—, æt. 56. Married; five children. Vaginal hysterectomy, August, 1901.

In order that the facts may be readily appreciated I have represented them in the subjoined table in so far as the age liability and parity is concerned.

TABLE I.

No.	Age.	Pregnancy.
1	56	0
2	50	0 Fib.
3	42	0
4	36	0
5	45	0
6	60	0 Fib.
7	54	1 Fib.
8	53	6
9	41	1
10	54	0 Fib.
11	56	5

To still further test these points, I asked Dr. Crewdson Thomas to tabulate the cases of cancer of the body of the uterus admitted into the Chelsea Hospital for Women during the years 1897—1900. The cases are represented in Table II, and the close agreement, in so far as age and parity are concerned, and in the co-existence of fibroids, is very close.

TABLE II.

No.	Age.	Pregnancy.
1 ...	43 ...	o Fib.
2 ...	41 ...	o
3 ...	55 ...	o Fib.
4 ...	54 ...	2
5 ...	62 ...	o
6 ...	43 ...	o
7 ...	60 ...	o
8 ...	60 ...	5
9 ...	60 ...	o
10 ...	65 ...	o

ON LATENT FIBROIDS.

Botanists apply the adjective latent to buds which remain undeveloped or dormant for a long time but may at length grow. It is precisely in this sense that the word "latent" will be used in relation to fibroids of the uterus in this article, the object of which is to point out the significance of latent fibroids in regard to the operative treatment of such tumours; it will, however, be useful at the outset to justify the title.

Let anyone take the trouble to examine a number of uteri obtained from individuals between the twenty-fifth and the fiftieth years of life, by the simple means of sectioning them with a knife, he will, in a very large proportion of the specimens, find small rounded bodies resembling knots in wood more than anything else, their whiteness being in strong contrast to the redness of the muscle tissue in which they are embedded. These discrete bodies, in many instances no larger than mustard seed, are embryonic fibroids, and in their histologic structure are identical with the fully grown tumour. An investigation of this kind is very useful, for the observer quickly realises that a uterus may contain many of these small bodies (ten or more) without the least distortion of contour or alteration in its size,—indeed, nothing to indicate to sight or touch that the organ contained anything abnormal. These small fibroids may never cause trouble, may never pass beyond this stage, and in

women who have attained the ages of seventy and even eighty years such minute fibroids may be detected, though in many instances they have undergone calcification. When a uterus contains many fibroids they are seen of all sizes; one or two may weigh several pounds, others a few ounces, but a careful examination will invariably reveal some no bigger than a mustard seed. A careful consideration of the great frequency of seedling fibroids, and their multiplicity when compared with the number of fibroids which attain proportions sufficient to render them clinically appreciable, makes it undeniable that an enormous proportion of them remain latent. That they may appropriately be compared to latent buds is shown by the fact that they may remain dormant through a long life, or assume active growth and become formidable tumours.

It is not an uncommon experience for an operator to dilate the uterine canal and abstract two or more submucous fibroids. However carefully the procedure be conducted, and no matter the thoroughness with which the walls of the cavity are examined for minute fibroids, no honest assurance can be given to the patient that other fibroids will not grow in her uterus.

A careful study of cases in which this has happened is instructive, as it affords some information as to the rate at which fibroids grow.

In April, 1901, I saw a patient æt. 40 with Dr. Gavin Stiell, on account of severe menorrhagia due to fibroids. Dr. Horrocks had enucleated a fibroid from her uterus six years previously, which was probably of some size, as it was extracted piecemeal. The husband was disappointed at the recrudescence of the fibroids, and wished me to remove the uterus. I was anxious to avoid this if possible, and succeeded in removing through the vagina six fibroids, two as big as acorns, three of the size of walnuts, and one as large as a bantam's egg. From what we know of fibroids the rate of growth in this instance was slow, even making allowance for the fact that the tumours in this case were of the very hard variety. In my book on 'Tumours' (second edition) I have briefly described a case in which I enucleated from the uterus through an abdominal incision a fibroid measuring 15 cm. in its major, and 5 cm. in its minor axis. The patient was twenty-three years of age, and mother of one child. She was delivered of a second baby at full time eight months after

the operation, so that it is reasonable to believe that she was pregnant at the time of the operation. Three years later she again came under my care on account of a pelvic tumour, and this grew so rapidly that I performed hysterectomy three months later, and found the uterus occupied by twenty fibroids, varying in size from a ripe currant to a hen's egg. The largest tumour occupied the cervix (Fig. 9). There were no signs of these



FIG. 9.—Uterus in section; it contained twenty fibroids, which grew in three years. The shaded parts were removed by operation. The largest tumour occupied the cervix, and its outline is introduced to complete the contour of the uterus. (From the Author's work on 'Tumours,' 2nd edit.)

tumours when the first operation was performed three years previously.

The tumours in this case attained a larger size in three years than those in the preceding case had reached in six years. This may be explained in

two ways. The patient was younger and had been pregnant since the primary operation, and I feel satisfied, from observations on similar cases, that *pregnancy exerts a quickening influence on latent fibroids*.

In June, 1901, a woman æt. 39 came under my observation with a large abdominal tumour. She stated that Mr. Meredith had removed a large fibroid from her abdomen. This surgeon kindly informed me in a letter that he removed from this patient in November, 1895, an extremely hard multiple fibro-myoma of the size of a large foetal head; it was impacted and very firmly adherent in



FIG. 10.—A uterus with fibroids seen in sagittal section. The large tumour in the posterior wall was myxomatous, and had grown in five years.

the pelvis, with a coil of adherent bowel (sigmoid) overlying it. The uterine connection was a thick fleshy pedicle springing from the left anterior aspect of the fundus. This was dealt with by ligature. The uterus was normal as to size and consistence—no trace of other growths being discoverable by palpation.

When she came under my care the crown of the tumour reached the level of the umbilicus, and it was extremely tender when the hand was placed on the abdomen; the woman also complained of profuse menorrhagia. I came to the conclusion that

it was a uterine fibroid undergoing secondary changes. Since the primary operation she had been living at Mafeking, South Africa, and had come to London to have the tumour removed.

On opening the abdomen I found the uterus uniformly enlarged and of a deep purple; it bled freely from any slight abrasion. Moreover its surface presented a peculiar villous appearance due to a multitude of short single tags of organised lymph. There were no adhesions except on the anterior face of the uterus near the right cornu, where it was in firm union with the lower angle of the abdominal cicatrix, the result of Mr. Meredith's operation. I performed what was practically a pan-hysterectomy, and removed both ovaries and tubes. The bleeding was free because the manipulations were somewhat hampered by the extreme fatness of the abdominal walls and the markedly funnel-shaped features of the pelvis. The patient recovered quickly and satisfactorily.

The uterus contained several fibroids, but the chief one was of the submucous variety, and lodged in the posterior wall. It was quite soft and of the myxomatous type. At first I thought the softening was due to infection, but the microscope did not support this view. It is well known that these soft (myxomatous) fibroids not only grow more quickly than the hard kinds, but they are associated with profuse menorrhagia. It is also an important fact to bear in mind that after enucleation they sometimes quickly recur.

Up to this date I have enucleated fibroids from the uterus on very many occasions. In four of the patients there has been a recrudescence of the fibroids which has necessitated a second operation. In two instances I have had to deal with recrudescent fibroids where other operators removed the primary tumour; these form the subject of this communication.

That similar cases have occurred to other workers there can be no doubt, but there is very little reliable information available to enable an estimate of their frequency to be even guessed at; under certain conditions, which easily suggest themselves to the minds of those who are engaged in performing these operations, this may have a very important bearing when an operator is deciding whether to be content to enucleate a uterine fibroid, or whether it is in the best interests of the patient to remove the uterus.

ON FIBROIDS IN A UNICORN UTERUS.

Several examples have been observed and recorded in which a fibroid has developed in the rudimentary horn of a so-called unicorn uterus, but in none of the cases did the tumour exceed the dimension of a potato of average size.

The subject of the present communication was a sterile married woman *æt.* 44. Some months before she came under my observation she sought advice in South Africa for a pelvic tumour, which was regarded as an ovarian cyst. The patient came to London for the purpose of having this tumour removed. On careful examination I satisfied myself that it was in all probability a uterus containing fibroids firmly impacted in the pelvis. It did not interfere with the bladder in any obvious manner, and menstruation was not disturbed. As far as I could interpret the facts, the pain and discomfort associated with the tumour seemed to depend upon the impaction.

The tumour was exposed by a fine incision in the middle line of the abdomen, and the uterus was seen to be occupied by several fibroids. The right ovary, twice its usual size, was found firmly adherent to the pelvic structures, and also to the cæcum. An unsuccessful search was then made for the left ovary. In order to facilitate matters I secured the vessels at the right uterine cornu with forceps, and removed the uterus and its cervix (pan-hysterectomy); then I could easily remove the enlarged right ovary and Fallopian tube from its bed of adhesions. A careful search was made for the left ovary and tube, but without success; the corresponding round ligament of the uterus could not be recognised. This led me to examine the kidneys, but both these organs were natural in size and position. After a repeated thorough examination of the pelvis, Dr. Comyns Berkeley, who helped me at the operation, agreed with me that the left ovary, tube, and round ligament were absent; the abdominal incision was sutured in triple layers in the usual manner. Recovery followed quickly and satisfactorily.

The subsequent examination of the uterus justified the view we took during the operation. The uterus (Fig. 11) is deformed by fibroids, and the right ovary with the Fallopian tube and round ligament are in their respective positions, but the ovary is nearly twice its proper size. There is no

trace of the left ovary, tube, or round ligament, but there is a left uterine artery. I then divided the

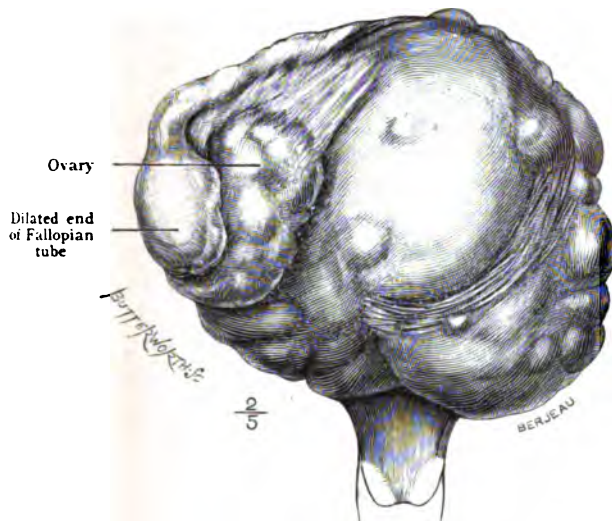


FIG. 11.—Unicorn uterus deformed by fibroids.

uterus in its sagittal axis, and conducted a very critical examination at what would correspond to



FIG. 12.—The uterus shown in Fig. 11 shown in sagittal section. The drawing shows the uterus in acute retroflexion, due to the fibroids on the posterior wall.

the left cornu, but there was no evidence of a Fallopian adit.

The study of the cut face of this uterus is interesting (Fig. 12). The anterior and posterior walls are occupied by fibroids, the largest tumour is situated in the posterior wall, and has so displaced the uterus that the fibroids in the anterior wall are directed upwards. The result of this displacement has been to cause the cavity of the uterus to lie at nearly a right angle with the cervical canal. When the uterus is distorted and displaced in this manner, the fibroid in the posterior wall will slowly grow and occupy all the available space in the true pelvis, and become so firmly impacted that it will often require a considerable effort on the part of the operator to extract it in the course of an abdominal hysterectomy.

ON AN ECHINOCOCCUS CYST (HYDATID) OF THE OVARY.

A careful examination of the relations which an echinococcus cyst bears to the colon, kidney, uterus, or spleen will soon convince the observer that the cysts in the first instance are deposited in the subserous connective tissue, and that when they invade the tissue proper of these organs it is secondary and due to the gradual increase in size of the cysts. I was interested in this matter about ten years ago in regard to echinococcus cysts of the ovary, for a critical investigation of the few available museum specimens and a study of the recorded cases led me to state in the second edition of my book on 'Diseases of the Ovaries' that "a primary echinococcus cyst or colony in the ovary is unknown," and curiously enough the testicle seems to be equally immune. It is true that Neisser in his industrious and oft-quoted paper ("Die Echinococcen Krankheit," Berlin, 1877) collected references of seven cases of hydatid cysts of the ovary; but an examination of the original reports shows that there was little reason in most of the cases to class them with hydatids. Indeed, one of the cases was an ordinary multilocular ovarian cyst. Even in the examples recorded in recent years now that the term "hydatid" is almost restricted to the true echinococcus cyst the cases recorded as "hydatid of the ovary" are conditions where the colony has grown primarily in the mesometrium, and implicated the ovary secondarily. Even the specimen which forms the subject of this communication can be included in this criticism.

A woman æt. 44 came under my care with what appeared to be a uterine fibroid as big as a foetal head at term impacted in the pelvis; this was associated with inordinate menstruation, but scarcely warranting the term menorrhagia. In addition to the impacted tumour there was a freely movable stalked tumour, as big as a turkey's egg, lying mainly in the right iliac fossa, but it could be pushed easily across the middle line. This was the chief source of the patient's trouble, for whenever anything pressed it the patient complained of acute pain. Two views were held in regard to this thing; the more obvious held it to be a pedunculated subserous fibroid, the other a small ovarian cyst.

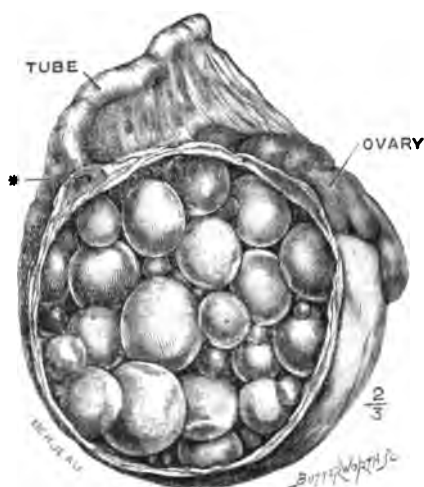


FIG. 13.—A mesosalpinx with the tube and ovary in transverse section. The ovary is flattened upon the wall of an echinococcus colony occupying the mesosalpinx. * The cut surface of the Fallopian tube.

At the operation it was deep red, elastic, and seemed like a pedunculated fibroid, but on examining its relation to the Fallopian tube it clearly replaced the right ovary. I removed it, and the impacted fibroid in the uterus was enucleated. On incising the elastic cystic swelling after its removal, we were all surprised to find it an echinococcus colony. Subsequently a careful examination of the parts was made, and the relation of the colony to the ovary demonstrated. The cyst itself is situated between the layers of the mesosalpinx, and occupies exactly the same position, and has the Fallopian tube stretched over it in the same fashion as a parovarian cyst. The ovary is flattened

out by the pressure of the cyst. The relationship of the parts is shown in Fig. 13.

It is an interesting feature of the specimen that there were no other echinococcus cysts obvious in the pelvis. In nearly all the cases recently reported as hydatids of the ovary there were colonies in the adjacent mesometrium, and in many of them the colonies supposed to be in the ovary were not merely contiguous, but continuous with those in the pelvis. The patient has been under observation many months since the operation, and there has been nothing to indicate the existence of other echinococcus cysts.

I am convinced that an echinococcus cyst in such close contact with the ovary, isolated and solitary as this specimen, is a thing of very great rarity.

A CLINICAL LECTURE MAINLY ON RINGWORM.

Delivered at the Hospital for Diseases of the Skin,
Blackfriars,

By PHINEAS S. ABRAHAM, M.D., F.R.C.S.I.,
Surgeon to the Hospital.

GENTLEMEN,—I will begin by showing you a series of cases.

1. The first is a patient with Bazin's disease, which was first described by Bazin many years ago as "erythema induratum" of the scrofulous. It is a condition which is now often recognised in this country. The lesions first come as raised bluish-red lumps on the legs, sometimes on the shins, more often on the calves. These may go on to ulceration, and very often form punched-out ulcers, which scar over in course of time. In former times they were often looked upon in this country as the lesions of congenital syphilis. This patient had the affection many years ago, and you can now see only scars. She has been attending lately for acne. There is sometimes in these cases a history of rheumatism, and also more often one of tubercle.

2. Here is a case of tinea versicolor. You see brownish patches, which are massed together on the abdomen and chest and shoulders, and scattered

over the back also. It is undoubtedly due to a fungoid growth. It is not always easy to get the fungus if the case has been treated; but we will try to do so by scraping the surface, and mounting on a slide with Liq. Potassæ. The affection is due to the fungus "*Microsporon furfur*," which grows in the epidermis and between the deeper layers. It can be cured pretty easily by the application of hyposulphites in solution, and germicide ointments. Phthisical patients are particularly liable to be affected, the reason being that they perspire very much. It is also apt to occur in people who use flannel next to the skin. I always recommend the wearing of smooth garments next to the skin. An old and common name for the affection was "pityriasis versicolor." A lady was brought to me in consultation the other day who had had it extensively for six or eight years; she had been under prolonged treatment with large doses of arsenic internally, but it does not seem to have occurred to anybody to scrape the surface and find the fungus!

3. The next case is one of seborrhœic eczema of a psoriasiform character. The condition is rapidly disappearing under treatment, though it existed for two and a half years. There are very small patches scattered over the front and back of the chest and arms. He is using a combined sulphur ointment after bathing with tar lotion.

4. This boy came yesterday with patches looking very much like *tinea circinata* about his arms and various other places. We scraped, but could not find any fungus. It probably is a case of *pityriasis rosea*, which generally appears first like a *tinea* patch on the chest; after a time an eruption of similar patches breaks out in the neighbourhood, and gradually spreads downward. There is usually some initial digestion disturbance. The affection tends to disappear spontaneously.

5. This boy came here yesterday with a crusted patch on the face, but there was something about the look of the condition which made me search for a fungus. I found that it was really a *tinea circinata*, in spite of its appearance of *impetigo contagiosa*. You can see under the microscope that there are distinct mycelia. It began with a pin-point spot, and then gradually got larger; now it is the size of a sixpence. He complains of pain on the left side of the face, where the spot was. No other children of the family have had ringworm. I have ordered him Ung. Hyd. Ammon.

6. Here is a girl with *tinea tonsurans*. According to the history she has only had it seven weeks. On examination we found abundant spores. There are short irregular hairs, not growing properly. If you see hairs in a child's head not growing in the right direction, you may always suspect *tinea tonsurans*.

7. This child came to the West London Hospital some time ago with an enormous patch of kerion, which was raised and swollen and perfectly bald. It shows that even if you have extensive inflammation with boggy thickening of the scalp, you may have the hair coming back after treatment.

Ringworm, of which you have seen several cases this afternoon, is a disease due to a fungus. It was not known to be due to a fungus until the year 1843. Before that time many people thought it was a kind of herpes or lichen mixed up with eczema. Gruby, a French observer, in 1843 found microscopically that there were fungi in and about the hairs of these cases. But he rather confused matters by calling the disease not ringworm, but "*porrigo decalvans*," another name for *alopecia areata*, which is a very different thing. Two years after that a Swedish observer, Malmsten, independently found a fungus in ringworm which he called "*Trichophyton tonsurans*." It was then at last believed to be a contagious disease, and inoculation experiments were made proving it to be so. It was also soon shown that the affection was closely connected with ringworm of the body, the so-called "*tinea circinata*," in which a fungus was also found. Until 1893 it was thought that there was only one fungus causing all the different forms of ringworm, of both scalp and body. In that year M. Saboureaux, a French dermatologist at the Hospital St. Louis, came to the conclusion that there were several different kinds of fungi, all belonging to the one family of saprophytes, but distinct in many respects, both in the methods of their growth and in the lesions which they produced. He classified them, broadly, into two great groups, the large-spored and the small-spored. He came to the conclusion that the majority of cases of ringworm were due to the small-spore variety, namely, about two thirds of the cases in France. He arrived at the conclusion; also, that there were several different forms of small-spored as well as of the large-spored fungus. In 1893 he sent over some specimens to me, and they are here now on the table. They

show the growths of some of these fungi. His observations have been repeated and to a great extent confirmed, and his views are now accepted in their broad outlines. Certain statements which he made have not been quite proved, but in the main his conclusions have been shown to be correct.

We all admit now that there are two distinct forms of ringworm fungi, a large-spored and a small-spored, the one being called "*Trichophyton megalosporon*," and the other identical with the fungus "*Microsporon Audouini*," first described by Gruby. I have here some cultivations for your inspection, which have been prepared by Mr. Pernet, the pathologist to the hospital. The tubes show the characteristic growth of certain forms of ringworm, viz. the "*Trichophyton megalosporon ectothrix*," and the "*Trichophyton megalosporon endothrix*." The ectothrix is a large-spored fungus which occurs chiefly in the sheaths and outside the hairs; one not infrequently finds this in ringworm of the beard. In the endothrix the mycelium of the fungus extends chiefly into the substance of the hairs. That hard and fast distinction, however, is now being somewhat doubted. The whole subject yet requires to be thoroughly worked out, but the broad lines as I have indicated are quite clear. Most of the ringworms of the large-spored variety come from animals. It is possibly due to that fact that in this hospital, long before Saboureaux's investigations, Mr. Tay pointed out to me large-spored ringworms; but we were under the impression that, as they chiefly occurred in the spring, the larger size of the elements was due to their more succulent growth in the warm weather. Possibly the explanation of the megalosporon appearing more abundantly in the spring is that children have then more to do with animals than at other seasons.

With regard to the treatment of ringworm, one has to try to destroy the fungus, and to render the soil sterile for its growth. That is quite an easy matter if the fungus is superficial, or not far below the surface of the skin. Anything will do—tincture of iodine, turpentine, sulphurous acid, or even common ink. Thus it is that tinea circinata of the body is so easily eradicated, for there are then no deep hair-follicles to be invaded. But tinea of the scalp is not always so easily cured, unless it is attacked at the very beginning. If the fungus gets deeply into the larger hair-follicles, the germicide which you apply may not reach it,—indeed, it is

often most difficult to get at it. Several methods of dealing with these cases have been tried, and one of them was to extract all the grease from the follicle by means of chloroform and ether, and then to apply a germicide dissolved in a similar medium. But even that method does not seem to be of much use when the fungus happens to be deep down in the hair-follicle. After a good deal of thought some years ago, and remembering the way that logs of wood for railway sleepers, etc., are creasoted, it occurred to me that one might get these remedies deeply into the scalp by a mechanical method, namely, by exhausting air from the follicles of the scalp, and then allowing a penetrating germicide to flow in under the atmospheric pressure. This is carried out by the instrument or pump I show you, and the use of which I can now demonstrate on one of these patients. I have been using this apparatus for obstinate cases for some years, and with considerable success; indeed, cases of ringworm of five or six years' duration have yielded in two or three months to this treatment. I am at present employing a mixture of creasote with tincture and liniment of iodine. As many hairs as possible should be at first epilated, and the scalp should be washed with hot water and soft soap, and then with alcohol and ether, to remove as much fatty matter as possible. It is advantageous to treat the cases with this machine two or three times a week.

In addition to this, as a routine treatment, I instruct the friends to rub in with a stiff brush night and morning an ointment containing one drachm of carbolic acid and one drachm of salicylic acid to the ounce of vaseline. These fungi require air for their development, and if you could prevent air from getting into the scalp the fungus would not grow. The ointment to a certain extent keeps air from them, and also prevents the fungus from getting distributed. I recommend that a portion of the same ointment be rubbed over the rest of the scalp, to protect the other parts from invasion. I allow the scalp to be washed twice a week with soft soap containing some antiseptic,—for instance, carbolic acid and boric acid, a drachm of each to two ounces of soft soap; and immediately after washing, the machine can be used for the patches, and then the ointment thoroughly applied.

One very common method of treating these conditions is by mercurial ointments. But the

use of strong mercurials is apt to produce mercurial poisoning. There was a case years ago in which a perchloride of mercury ointment was used, and as a result of it the child died. Chrysarobin has also been used in these cases. It is certainly a very good germicide, but its drawback is that it stains linen very badly, and it is also apt to produce erythema of the eyes and face. On the whole we get fair results by the above salicylic and carbolic acid ointment. I generally insist that the scalp be shaved once a fortnight, so that it can be better seen if there is any spreading of the condition.

Sulphur in the treatment of ringworm has been very much lauded, but I cannot say that I have had good results from it. When formalin was first brought under notice as an agent for hardening and preserving museum specimens, I thought it might be useful for ringworm, and I tried it in about thirty cases. I found it, however, to be such a terrible escharotic that I could not use it in any strong form. Even of a strength of one in ten it produced a great deal of painful dermatitis. Some time afterwards I was surprised to find it brought forward as a certain cure for ringworm. I can only say that a good many cases of active ringworm have come under my notice after their supposed "cure" by means of formalin. That formalin will destroy the fungus I do not deny, but it will also at the same time destroy the epithelial cells and tissues around. Many cases have come here with great scarring due to the use of the formalin, and yet with the ringworm not eradicated. Another agent which has been for long lauded for tinæ tonsurans is croton oil, the idea being to produce an inflammation in the follicle, which will form pus and extrude any fungus which may be there. Some authorities are still using it, mixed with olive oil, by means of a fine steel probe passed into each diseased follicle. This method obviously involves an enormous amount of trouble and care, for there may be hundreds of follicles to treat in one patch, and it is admitted that even under such treatment a case may last for years.

No method is indeed infallible; some cases resist treatment in a most remarkable way. I must maintain, however, that in my hands the method I have shown you is the most efficacious that I am aware of in old long-standing cases.

It is important not to discharge a case until

every trace of the condition is gone; treatment must be continued if there is the least degree of scurfiness left, or if a single stumpy hair can be found.

I need hardly remind you that the microscope is a most important adjunct in the diagnosis of ringworm. Children very often come to this hospital with a generalised scurfy condition of the scalp, which has lasted, and has often been treated as eczema, for years. Patches of typical ringworm may, indeed, become merged together, and the whole scalp become covered with scales so thickly that the short hairs are scarcely or not at all visible; nothing but scurf to be seen. By scraping, the scales and the hidden short hairs can be examined in Liq. Potassæ, and you will find the fungus in abundance. Sometimes, in fact, one cannot be certain of the diagnosis of a case without the aid of the microscope.

An important question in London is the prevalence of ringworm in schools. The majority of the subjects of the condition who come under my notice here, and at the West London Hospital, have contracted their ringworm in the board schools. I think the time has come to do something in the matter. It is not fair that poor people should be forced, as they are, to send their healthy children to institutions where there is every probability of those little ones contracting such diseases. Under the present system infected children may and often do attend school for weeks or months, and spread ringworm, pediculosis, etc., unchecked. The obvious remedies are that, in the first place every school be periodically inspected by a skilled medical observer, and secondly, that all cases of ringworm be isolated from healthy children, and taught in separate rooms from the others, or better still, in distinct and special schools. If school boards would spend some of the ratepayers' money in this direction, rather than as they do at present, they would be doing their duty, and there would, at any rate, be less just cause for complaint.

**WITH DR. CRAWFURD
IN TWINING WARD,
KING'S COLLEGE HOSPITAL.**

THIS boy seems to be suffering from the "dry" form of chronic tuberculous peritonitis. You could not have a more typical clinical picture to carry in your mind's eye. There is the usual history that for some time he had been ailing and losing flesh until two months ago, when a sudden increase in severity of the symptoms, probably due to a fresh outburst of tuberculosis, led to his case being diagnosed as one of typhoid fever. Now he comes to us a mere skeleton; his temperature is typically hectic; he is never free from uneasiness or pain in his abdomen. His abdomen is distended, and on its lower part the branching veins are as visible as if they had been marked in with a blue pencil. It may be that they are somewhat distended, but they are chiefly shown up by the absence of subcutaneous fat. The abdomen is now hard as a board, and very tender on pressure. A week or two back one could readily make out several irregular swellings in the abdomen, all of which were resonant on percussion, and represented matted loops of distended intestine. This condition, and the liability to vary from time to time, are very characteristic of tuberculous peritonitis. I cannot detect any fluid, but it is never safe to say that there is none, for when shut in by adhesions it may not shift with gravity, and it may have resonant bowel in front of it. Such other symptoms as he presents are just what one would expect in a patient whose bowels are hindered in every attempt at movement by widespread adhesions. Appetite he has none, except a perverse desire for shrimps and raw apples, and this is one great difficulty in treating him; the simplest food provokes pain. Constipation is giving less trouble than is often the case.

One used to be taught that tuberculous peritonitis was probably never a primary condition, and that some other focus of infection might always be found either before or after death. In this case it is true there is evidence of a quiescent process—probably tuberculous—at the right apex, where you see there is flattening and limitation of movement. But it is now generally admitted that the peritoneum may be affected by the passage of bacilli from the

intestine without any lesion of the intestinal mucous membrane, and with very slight, if any affection of the mesenteric glands.

At present the boy is far too weak and ill to consider the question of laparotomy. We are simply feeding him with as much simple food as we can get him to take, and giving him iodoform in pill by the mouth, and iodoform inunction to the abdomen. If you care to test the saliva during iodoform inunction you can demonstrate to yourself that the drug is readily and rapidly absorbed. This is not a very favourable case for medicinal measures, as there is no free fluid in the abdomen. Why these latter cases should do best I don't know, but perhaps the fluid is a means of bringing the antiseptic iodine into prolonged contact with the tubercles. He has certainly made some progress, and I hope that soon we may be able to consider the question of laparotomy. I do not consider the pulmonary lesion a serious contra-indication, as it appears to me to be arrested. We know nothing of the *rationale* of laparotomy in tuberculous peritonitis. It has been suggested that the good result is due to removal of the fluid—and it is true that cases with fluid are more amenable to surgery as well as to medicine,—but the objection to this view is that many cases of the "dry" form are also cured by operation. Then again, the admission of light has been suggested. It is a pleasing fancy—these cave-dwelling tubercles dazzled to death by the admission of a ray of light into the nethermost gloom of the abdomen,—but if you have ever watched a laparotomy in an advanced case, and perhaps remarked on the difficulty of the surgeon in getting even the tip of his finger into the peritoneum, you will probably discount heavily the value of light. Others have championed the admission of air; this is difficult to prove or disprove, but this we do know that the tubercle bacillus is *aerobic*. Nor is it the use of antiseptics, for most surgeons will tell you that they have given up flushing or sponging the abdomen, and are content simply to evacuate any fluid and close up the abdomen. I fancy the natural tendency of every tubercle to obsolescence must in some way be strengthened by the abrupt change in the capillary circulation of the peritoneum that must follow the sudden alteration of intra-abdominal pressure.

In this next bed we have a case of cirrhosis of the liver with ascites. 'Ασκίς, as you know, means

a wine-skin, and if you look at the man's abdomen, you will see that it is full of fluid. And he is a wine-skin in another sense, for there is a history of prolonged alcoholism, which is almost without exception always the cause of cirrhosis. It is worth contrasting his appearance with that of the boy we have just seen. Like him he is emaciated, but not to the same extent; the abdomen is enormously distended, and the bagging in the flanks tells you at a glance that you have to do with free fluid. The pressure has also bulged out the navel, so that it forms a small hernial protrusion. On the lower abdomen the veins are marked, but not so obviously as in the last case, for though perhaps more obstructed, they have a better covering of subcutaneous fat. If you put your hand on the abdomen you will find the wall comparatively flaccid, or at any rate quite free from rigidity, and on percussion there is uniform dullness, but for a zone around and above the navel. We have not been able to feel his liver even after tapping, and I presume that it is shrunken in size. The spleen is enlarged, and he has troublesome piles. He has never had hæmatemesis. His urine contains albumen, and some hyaline and epithelial casts, so that he probably has kidney disease as well, as is often the case. A few days ago his scrotum was enormously swollen, and boggy and angry looking; this was most likely due to the pressure of the fluid in the abdomen on the veins and lymphatics, and has subsided now that the scrotum is braced up in a sling. Dyspnoea has already compelled us to tap his abdomen on several occasions, but in the course of a week the abdomen becomes as full as ever. Students are apt to have quite a wrong picture of cirrhosis. The evidences of portal stasis—the dyspepsia, hæmatemesis, the enlarged spleen and abdominal veins, the piles, and the ascites—are so prominent in the foreground that one is apt to lose sight of the essential toxæmia in the background. It is as if, in cirrhosis of the kidney, one were to fix one's attention only on the condition of the urine and the state of the circulation, and put out of mind the existence of uræmia. Chronic peritonitis is almost as much a part of most cases of cirrhosis of the liver, as arteriosclerosis is of cirrhosis of the kidney; and as in cirrhosis of the kidney uræmia may be the first indication of its existence, so the onset of coma may be the first evidence of cirrhosis of the liver,

when the signs of portal back-pressure are absent. I spoke of ascites as an evidence of portal stasis, but this is far from being the whole truth. Ligature of the portal vein does not cause ascites, so that when ascites comes on in a case of cirrhosis of the liver, you may be sure you have to deal with something more than simple back-pressure. That other factor is usually either chronic peritonitis or the irritation of the peritoneal capillaries by a toxin in the blood. In the former case the ascites is apt to be persistent and, as in this patient, to require repeated tapping; in the latter case the ascites is merely one evidence of an inevitably fatal toxæmia, and may require no relief, or at any rate not repeated relief. It is the same with hepatic ascites as with renal dropsy: pressure has very little to do with it, and poison very much. The hopeless corollary of all this is, that however successful we may be in tiding over the time till his collateral circulation is established, the evil day must yet inevitably come, when the toxæmia will claim its victim. Recently, as you know, operative measures have been adopted for cirrhosis of the liver. The idea has been, by producing perihepatic adhesions, to form new vascular channels for the return of blood from the portal area. The operation has for the most part failed of this effect, and in any case it ignores the very essence of the disease—the toxæmia. It has, however, been soberly contended that the increased vascularity may bring about such a hypertrophy of the healthy residue of liver as to nullify the toxæmia.

This next man is just relieved of a very obstinate sciatica. A few years of out-patient practice, I think, justify one in forming one's own views on sciatica, without regard for authority, I mentally divide the sufferers into two main groups, those who have sciatica, and those who think they have sciatica, and this latter group I assure you is a very large one. This man undoubtedly has had a true perineuritis; its obstinate and unvarying character pointed to that. In chronic cases such as this it is mere waste of time to expect relief from local anodynes, or from drugs that are credited with the power of altering morbid constitutional states. The only course is to put the patient to bed at once, and apply the cautery pretty liberally along the painful area of the nerve. In this case we got almost complete relief after a single such application, but after ten days there was some slight

recurrence of pain. This I am glad to say yielded readily to massage and galvanism of the limb, used on alternate days for a week. A word or two as to the other group, those who think they have sciatica. I mean that functional pain—call it neuralgia if you please—in the sciatic nerve not only has an existence, which several modern textbooks deny it, but is also extremely common. There are many people who, after a hard day of physical exertion, experience discomfort along one or both sciatic nerves, which is relieved by a night's rest in bed; are we to say this was neuritis? or are we to deny that it was sciatica? I have met with sciatica in a young lady who had been bed-ridden for five years and had had the best treatment of the most experienced London physicians, that yielded in one sitting to hypnotic suggestion. And one comes across many cases of mild sciatic pain that yield quite readily to local anodynes. I have a very strong suspicion that sciatica is sometimes reflexly excited by an aching varicocele, and in two instances I have seen a mild but chronic sciatica disappear on removal of a varicocele. And once or twice I have seen sciatica so mixed up with the trouble of an enlarged prostate, that I have wondered whether the prostate has not excited a sympathetic ache in the sciatic nerve.

In the next bed is a man with malignant disease of the bladder. The past history in a nutshell is hæmaturia for more than a twelvemonth, and aching pain, perineal and suprapubic, for the last six months. As late as five months ago his bladder was examined by cystoscope, and no cause found for the profuse and persistent hæmorrhage. Six weeks back he was brought by the police to the hospital unconscious, apparently from loss of blood into the bladder. He was then found to have a mass of growth in the inguinal glands; this mass has steadily increased in size. Per rectum the prostate is perfectly normal, and except under an anæsthetic, it is impossible to feel the extensive disease of the fundus of the bladder. Mr. Burghard has now examined him for me with the cystoscope, and reports that the whole inner surface of the bladder is the seat of extensive malignant ulceration. It is remarkable how easily he voids his urine—fortunately the prostate is absolutely free, so that I hope we may throughout have no trouble in emptying the bladder. To-day you see a quantity of pus in the specimen of urine; yester-

day it was almost all blood. Of course, when the surface of an ulcer ceases to bleed it will still throw off pus. We have tried to relieve his pain by morphia suppositories. To check the bleeding is a more difficult matter. We tried to wash out his bladder, and leave a solution of adrenalin chloride in contact with the ulcer, but the passage of even a soft catheter excited so free bleeding that we gave it up. I have thought of trying gelatine, either hypodermically or in normal saline solution by the rectum, but just at present gelatine is under a cloud.

This boy has, I believe, renal tuberculosis. The history is that for nine months he has been ailing and losing flesh, and has seldom gone more than a few days without pretty free hæmaturia; he has also had a constant gnawing pain, first in the left loin, now in his right. He has just lost a little sister of tuberculous meningitis. The first specimen of urine I have seen, a few weeks back, contained abundant blood; some pus; and some blood, epithelial, and hyaline casts. The blood casts indicate that the blood comes from the glandular substance, but the blood is far too profuse for chronic nephritis, though the epithelial casts show that there is some catarrh of the tubules; moreover the constant local pain and the pyuria indicate that there is something more. I cannot make out, certainly, any enlargement of the kidney, and if it were a sarcoma I should expect by this time to find a palpable tumour. The copious hæmaturia and the scanty pus suggest calculus, but the pain is not colicky nor worse on exertion, and the radiographer also says "No." One is almost driven by exclusion to tuberculosis, but we have failed to find bacilli in the sediment, and at present the inoculation test is also negative. The temperature is persistently normal, and does not help us at all.

A New Sign of Raynaud's Disease.—A. I. Pospeloff ('*Medicinskoie Obosrenie*' June, 1901) observed a large number of cases in which Raynaud's disease was associated with biting at the nails, and he therefore concludes that a symptomatic relation exists between the two. He explains this "bad habit" by the fact that in Raynaud's disease the circulation of the matrix of the nails is disturbed, exerting pressure on the nerve terminals. This produces unbearable pain and a sensation of fulness in the nails, compelling the persons thus afflicted to bite at the latter.—*Philadelphia Med. Journ.*, Sept. 21.

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* Specially reported for The Clinical Journal. Revised by the Author.

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THE RESPONSIBILITY OF THE ORGANISM IN DISEASE.

Being the Presidential Address delivered at the opening of the "129th" Session of the Medical Society of London.
By W. H. ALLCHIN, M.D., F.R.C.P., President.

FELLOWS OF THE MEDICAL SOCIETY AND GENTLEMEN,—Among the widest spread inclinations of the human mind, however uncultivated or however learned, is the desire to ascertain the cause of such phenomena as come under observation. Instinctively one seeks for the precedent condition to which may be attributed the event we are concerned with, and it is scarcely going too far to say that when once this is ascertained further interest in the subject not infrequently lapses. That this is the case in respect to disease, so far as the laity is concerned, is, I take it, well known to most of us, and our patients after the malady is named at once are concerned to inquire its cause, and are generally fully satisfied by ascribing it to "a chill," more or less real, or in finding some ancestor supposed to have similarly suffered. Although to the pathologist and physician such references are but partially convincing, it may be noted that these explanations typify the two main groups of conditions to which may be reduced the varying circumstances that prejudicially affect our well-being, contrasting, as they do, the individual and his inheritance on the one hand, and on the other the external conditions to which he is subjected.

At the risk of offering you no novelty, and of dealing with a subject that is largely conjectural in its bearings, though not entirely without practical application, I propose to occupy the occasion which pertains to the position you have summoned me to by offering some remarks upon that aspect of the subject which more particularly concerns the individual, and to consider how far he is himself responsible for any given condition of disease. And I may say that I have been somewhat led to this choice of subject by what, unless I am mis-

taken, appears to be an indication that in the far-reaching and all-important extension of our knowledge, by the discovery of the part played by microbes in the production of morbid states, that the personal factor is running the risk of being somewhat lost sight of, if not admittedly, at least in fact.

THE PRESENT CONCEPTION OF THE NATURE OF LIFE.

The conception of life as held at the present day, and expressed in terms that are consistent with our general notions of the cosmos, is that it is the sum total of the phenomena exhibited by certain bodies, resulting from the interaction between a special constituent of those bodies—bioplasm (cell)—and the environment. Thus stated there are clearly two factors to be considered, in the modifications of the one or other of which are to be found the immediate and predisposing causes of altered vitality, *i. e.* disease, whilst the resulting phenomena themselves, independent to some extent of the circumstances of their causation, afford a field of study that furnishes the various signs and symptoms of the different maladies we are called upon to investigate, and that should for any intelligent appreciation of them be compared with the corresponding manifestations that are taken as normal.

With the shifting point of view that increasing knowledge has furnished, now advancing as the knowledge is confirmed, now falling back as some false wind of doctrine has diverted inquiry from its true course, these two aspects of the subject have been variously credited with importance or the reverse, as being responsible for the causation of disease. The most superficial retrospect of the past in this respect will show how opposite are the views that have prevailed. In this, as in many other points of pathological doctrine, we find the earliest recorded Hippocratic teaching strangely similar to that of the present-day. There were the several 'temperaments' corresponding to the four fundamental 'humours,' a discrepancy in the mixture of which, and an undue preponderance of either would be the cause of disease, which clearly represents the responsibility of the individual; but equally did the Father of Medicine teach that disease might arise from external influences, such as air, season, climate, water, upon which he wrote largely. No such complete conception of the

ætiology of disease has existed until recent times erroneous as, of course, was much of the sub-structure. One or other factor was from time to time held as the all-important, though it would probably be correct to say that the most generally prevalent notions ascribed to the individual rather than to his surroundings, the determining circumstances of disease, and this notwithstanding those who taught the entrance *ab extra* of evil spirits, which brought about each its peculiar malady. The idea of disease as a correlative of health, and both as aspects of life, did not enter into the conceptions of the ancients. Disease was with them a something superadded to the body, whatever its origin, and the long-maintained doctrines of the humoralists, as also the very different doctrines of the vitalists in their numerous modifications, saw in flaws in the humours or fluids of the body, or in errors of a vital principle contained in the body, an explanation of disease. The dependence of function, whether healthy or morbid, upon structure, is of modern conception, and it is left still to us to discriminate how far the departures of the structure from a healthy standard are due to intrinsic, and how far to extrinsic conditions.

Although the main purport of my remarks is the share taken by the individual in the causation and manifestation of disease, yet it is necessary to an adequate understanding of my theme that I should say a few words upon the external conditions to which every living being is subject.

THE ENVIRONMENT.

In proceeding to consider the surrounding circumstances of the organism, which in some measure and degree are essential to its well-being, but that beyond those normal limits become sources of harm and the causes of so-called acquired disease, it must be remembered that the investigation of their action must not be limited to their effects, more or less obvious, upon the body *en masse*, but must extend further and include if possible the influence which they exert upon the component cells and tissues, and especially upon the bioplasm itself; and on this subject a large amount of experimental evidence has been accumulated.*

* 'Experimental Morphology,' by Dr. Davenport: Macmillan, 1897.

For present purposes the environmental factors may be thus grouped :

(i) Violent contact, represented by blows, injuries, and the laceration of internal structures by foreign bodies, such as calculi and the like. As the causes of maladies, arbitrarily termed surgical, since their treatment mainly involves manual skill, they are obvious ; but it is not to be forgotten that in infinitely less degree only, is mere contact a normal provocative of those metabolic changes which find expression in contraction, movement, and locomotion. Just as the loosely-associated molecules in those chemical compounds known as explosives are rearranged with great display of force when thrown into vibration, so parts, at least, of the highly complex and unstable substance bioplasm are similarly disturbed, with the manifestation, among other things, of energy termed contractile, nervous, and possibly secretory.

(ii) Another group of external conditions is represented by such natural agents as heat, light, and electricity, and as barometric pressure, gravity, and such subsidiary conditions as the relative humidity or dryness of the atmosphere. With extremes of any one or more we recognise the occurrence of disease in various forms, sometimes with no difficulty, at others only vaguely and with uncertainty, as for instance in the different degrees of malaise or positive illness that we associate with change of weather, itself a complex of these various agencies. But although there is much obscurity in detecting the exact effects that are produced upon the body as a whole by these ever-present conditions, in the innumerable combinations in which they occur, we have very precise knowledge concerning some of them in their separate action upon isolated living cells, and there is as little doubt that they may become responsible for disease as there is for the absolute dependence upon them of life, or the benefit they confer when employed as therapeutic agents.

(iii) A third set of conditions that are brought to bear upon the body and its component parts from outside are those which produce their effects in virtue of the chemical changes which they set up. Therein will be included modifications of the ingesta,—solid, liquid, and gaseous,—and perversions of the alterations they may undergo in the process of digestion or absorption, in fact, the whole class of substances denominated poisons, of whatever source.

As causes of disease these bodies are well known, and the recent development of bacteriology and a knowledge of the toxins elaborated by micro-organisms has widely extended our acquaintance with agents of this group, and consequently of our understanding of the ætiology of many maladies, perhaps at the risk of overlooking some of the other, and even more subtle and less understood agencies previously enumerated. Investigations into the mode of action of poisons upon living protoplasm show that they act by altering the minute structural arrangement or chemical constitution in various ways. Thus some determine an oxidation of the proteid, carbo-hydrate, or fat constituents of the protoplasm, as some act by substituting chemical elements or radicals for others in these components, and the general outcome is that the toxic properties of each substance are closely connected with the complexity and instability of its composition.

Although these several groups of agents are spoken of as external, that term is not to be taken as necessarily implying extrinsic to the entire organism, for in the inconceivably complicated structure of the higher forms of life, cells and groups of cells and tissues undoubtedly exert an influence beneficial or otherwise upon other cell-groups quite apart from what is occurring outside the body as a whole. Here it is that we should place the autogenetic poisons resulting from perversions of tissue metabolism ; and it is at least conceivable that influences, favourable or inimical, are mutually excited among bioplasmic units of a character which we cannot at present formulate, but which are familiar to us in their remote results, such as the effects of internal secretions, or of those peculiar forms of nervous stimuli which we designate trophic. Herein also would be included the consequences of the mechanical pressure exerted by new growths, inflammatory exudations and the like, upon adjacent tissues, which are manifested as degenerations, necrosis, etc.

In this extended view of the nature of the environments, the influence, that is, which may be exerted by cells and tissues of an organism upon other component cells and tissues of the same should lead us to realise the exact bearing which the so-called complications and sequelæ hold to the original malady. The perverted intonations which

ensue upon the infliction of the primary causes in their turn become disturbing factors in the life course of cells that are remote from the original seat of mischief, and lead to the manifestation of morbid symptoms that may altogether overshadow, both in importance and extent, those of the primary disease. Whilst, however, in the strict analysis of the condition the causation of these complications is to be found in the environment of the tissues affected, it is impossible, from the practical point of view, to regard them as other than those for which the individual is responsible, and to which the actual condition of the secondarily involved structures contributes not a little.

If we endeavour to penetrate still more closely into the way in which these several agencies affect the bioplasm, it is needful to remember the extraordinary complexity of this substance, which so eludes precise investigation of its composition, since it is altered and ceases to retain its living characteristics by the very means adopted for its analysis. Enough is however known to justify the view that its structure resembles that of a foam consisting of incomplete loculi formed by delicate strands and septa in the meshes of which the serous fluid, or at least diffuent material, is contained. Entering into the composition of this highly complex, profoundly unstable substance are proteid molecules, as well as fat and carbohydrate molecules and salts, the intimate chemical relations of which—the chemical structure so to say, for this, to my mind, is but the ultimate stage of its anatomy—are quite unknown. Now the action of the environment upon this material essentially consists in bringing about alterations in its structure and constitution, and with that affecting its functional capacity. Extreme conditions, such as great heat or cold, excessive electrical stimulation, etc., so affect its integrity as to determine a state of inaction, which is either suspended vitality or death according as the state be recoverable from or not. Gross molar injuries disturb with violence the complex structure; variations in degree of moisture by the relative amount of desiccation they produce; chemical agents by altering the essential composition of the bioplasm, and so interfering with the molecular atomic rearrangements which underly its exhibitions of energy; and light, heat, and electricity presumably act in a similar way. From the investigation of

this subject one or two facts are ascertainable that specially bear upon the subject I am now particularly considering. One is that bioplasm far from being a material uniform in composition and characters, exhibits very great variety, not only in the cells of different tissues, but in the same cell at different times and under different circumstances. Evidence of this is seen in the various behaviour of cells to staining reagents; and, indeed, different parts of the same cell respond to these different dyes in a different way. Still more striking, as showing the variable composition and constitution of bioplasm, are the effects of poisons, some of which are quite innocuous to some cells whilst violently toxic to others, and the greatest variability exists in the degree of resistance offered by different cells, whether they be unicellular organisms or the component cells of the tissues in the highest forms of life. Indeed the therapeutic value of many drugs—certainly of those whose use is based upon their known physiological action—depends upon the selective capacity of certain tissues as contrasted with others. A still more important point bearing upon this subject which has come out in the course of experiments on bioplasm, is that within certain limits the resistance of the living matter may be altered in such a way that agencies which ordinarily would be prejudicial to its activities, or even fatal, might be submitted to with impunity. This fact of adaptability to its surroundings, this ability to become acclimatised, so to say, we speak of as immunity when the agent is a toxic one. But the same results are to be found when the disturbing factor is heat, light, or electricity.

Another interesting phenomenon in connection with the effect of external conditions acting within certain limits upon bioplasm, is the determining of the direction of movement exhibited by living matter, upon which they exert an attractive or repellent effect—one form of this we are familiar with as chemiotaxis in states of leucocytosis, but it is probable that other forms of "taxis," caused by heat, light, etc., also prevail—for experiments with unicellular organisms and even some more complicated clearly suggest such to be the case.

Ultimately, no doubt, the mode of action of these agencies, whether this be in a direction favourable to the maintenance of the organism and to its satisfactory development, or as tending to affect it

to its harm, is a question of interference with metabolism, a term which is expressive of the constant interactions that take place between the protoplasm and its environment, and of the interactions among the constituents of the protoplasm. But inasmuch as one of the parties to this interaction is the living organism itself, which is of very variable nature and capable of artificial alterations in its behaviour, it is evident that the living matter is partly responsible for the result that any external agent, good or bad, produces. And, further, to be satisfied with merely ascertaining the external condition which has led to any condition of disease, is to take a very narrow view of ætiology; whilst to endeavour to throw the whole responsibility for the ultimate results upon this external condition is neither scientifically accurate nor practically sound. It can scarcely be urged in any such assembly as this, "What have we to do with bioplasm and cells, we want to recognise the signs and symptoms of the maladies we are called upon to deal with, and if possible to cure them—or at least to treat them." Inasmuch as the first object of our art is to prevent disease, and the final pursuit of pathological inquiry is to ascertain the precedent causal conditions, anything that assists in forming a more accurate conception of the nature of the various causes and their modes of action should be welcomed, even if the practical value be not apparent on the face of it. And whilst we at once accept poisons, extremes of heat and cold, and the like, as the causes of disease, it is in their action upon the living constituents of the tissues that they really produce their effects, and the more precise we can be in locating their results the more likely we are to be successful in applying their antidotes or in averting their consequences.

In attempting to comprehend therefore the effects of external agencies, however simple or however complicated they may be, we have also to reckon with the responsive capacity of the living organism. Thus it is that we see the various effects of the same untoward circumstances upon different individuals. How it is, for instance, that the exposure to cold and wet will in several persons similarly circumstanced bring about very different forms of disease; whilst the one develops an acute nephritis, another is attacked with acute pneumonia, another falls ill with what is known as a

rheumatic attack, and a fourth maybe suffers not at all. More precisely the differences exhibited by individuals in respect to natural immunity to infection, and still more strikingly the immunity that may be artificially acquired, and perhaps most impressive of all the various forms of response to particular foods, drugs, or other ingesta, known as idiosyncrasy, conclusively demonstrate that in modifications of the working of the organism it is not only the disturbing external factor that has to be considered, but also in some measure the organism itself. Great and important as was the discovery of the microbic cause of tuberculosis, I venture to think it will be long before those who have had any extensive experience of the ravages of that disease, will admit that the *Bacillus tuberculosis* is the only thing to be kept in view, either in the causation, the preventing, or the treating the malady, and I further presume to affirm that any teaching that fails to take account of the personal or intrinsic factor in the pathology of the disease is contrary to the light and likely to be harmful in its results

THE ORGANISM.

Whilst it is quite possible to investigate the phenomena and conditions which occur external to living things quite apart therefrom—indeed, vast branches of inquiry, such as physics, chemistry, astronomy, are entirely so occupied—it is impossible to deal with living things apart from their environment, nor is the attempt to imagine them so isolated in any way profitable. It is as the resultant of the interactions between a complex substance and the conditions around it that we find life, and with the cessation or grave disturbance of these interactions life ceases and death occurs. The consideration of the living organism therefore, apart from these conditions, is inconceivable.

Given a certain state of the living matter—a state quite indefinable—and a certain state of the environment, the resulting interactions are recognised as healthy, and these natural states are regarded as satisfactory. Variations in these states are possible, within limits, without any recognisable ill effects, but with departures from a certain standard of environment perverted interaction—disease—appears. This responsibility for disease on the part of the environment has just been considered, and the limitation of that responsibility

pointed out. It now remains to consider whether the organism itself can be said to have a similar responsibility; whether disease is ever wholly intrinsic in origin, or whether it is always dependent in some measure upon externals. In considering this aspect of the question, there would seem to be two directions in which an essentially intrinsic disease might originate, viz. as the result of inheritance, or as self-developed after birth; but in each case it resolves itself into an inquiry concerning the character of the material—bioplasm—through which life, whether healthy or morbid, is manifested, and its ability to transmit acquired modifications such as disease is.

There is one characteristic of living matter which, though quite obvious, is apt to be lost sight of, and although manifested under the influence of external conditions, cannot be regarded as essentially dependent upon them. We are in the habit of referring to the living elements of the tissues without due regard to their entirely transient nature. The peculiarly vital manifestations—the various forms of irritability in response to stimulation, the capacity for assimilation of extraneous material, the adaptability within certain limits to external conditions, and the power of reproduction—by which we recognise the presence of bioplasm, are continued until they naturally terminate in death. But the material itself through which these functions are manifested is ever changing, as the exhibition of these various forms of energy is the expression of a perpetual molecular disintegration and renewal. Thus the functional manifestations are continuous, though the underlying structural basis is constantly shifting; the muscle-fibre or nerve-cell of to-day is freshly constituted by tomorrow, though its capabilities remain constant. It is true that the *form* of the substance—the cell, the fibre, etc., persists, but not the actual constituents. We are bound to regard so remarkable a series of phenomena as a continued repetition of similar form with identity of potentiality, but with a perpetual change in material constitution as an inherent attribute of the living matter, for which the environment cannot be conceived to be primarily the cause, however much it may be subsequently involved; and surely the full appreciation of such a complex must suggest the directions in which the organism itself may become responsible for perverted functions, which we

recognise as disease. The substantial identity of life under the greatest diversity of environment gives further support to the same contention.

As with other questions of a kindred nature, opinion has undergone many changes at different times. The responsibility of ancestry for the occurrence of disease was, even recently, most extensively invoked. In its extreme application it constituted a form of fatalism and relieved the individual and his surroundings of all, or almost all, share in the causation of disease. It formed a convenient way of shifting on to our predecessors the responsibility for conditions the existence of which later knowledge has shown should have been more properly attributed to the environment. At the present day, with the vast impetus that has been given to the investigation of external conditions by the discovery of the bacterial origin of many diseases, there is to my mind a fear that the pendulum may swing too far in that direction—and indications of this, I think, are not wanting,—with the result that too little attention is paid to the individual, and the share he takes in the origination of the disease, or, what is more important, the part he should be made to play, whether in the prevention of disease, or in the treatment of it when existent.

That certain characters, structural and functional, are heritable is a matter of certainty. The offspring closely resembles the parent, though minor differences exist which exclude absolute identity; other differences occur among the offspring themselves, and equally exclude identity in their case. Moreover this resemblance of progeny to parent is anticipated and relied upon. No one expects a chestnut to grow from an acorn, nor a cat to be developed from the fertilised ovum of a bitch; anything but an oak or a dog respectively would be against universal experience. Nor is it for a moment suggested that this specific similarity of offspring and ancestor is determined by conditions of the environment, or that it is otherwise to be explained than the expression of the potentiality of the blended sexual elements; and the same would be admitted regarding the possession of characters, however trivial or unimportant, that would be clearly accepted as transmitted. But it is a highly disputed question how far, if at all, such somatic modifications as the individual may have acquired can be transmitted to his posterity. It might have been supposed that the innumerable perversions of

structure and function manifested in the many forms of disease with which we are acquainted, would have afforded the opportunity of determining this question, but it cannot be said that such is the case, and the subject of the hereditary transmission of acquired characters still divides biologists. The hypothesis of germinal continuity with which the name of Weissmann is especially associated, offers at least an explanation of a large number of facts, and what is of great value, affords a comprehensive and intelligible basis upon which thinkers can formulate their points of agreement and difference, and thus permit a common ground of argument, where previously all was vague, and much disputation was at cross purposes for want of an accepted definition of the subject. Briefly stated the hypothesis—and be it observed, it is little more than an hypothesis, at least so far as being universally applicable—is this :—The germ-plasm of the fertilised ovum, whilst in great part undergoing structural development and differentiation into the likeness of the parent form, remains in small part unaltered, and in some species, even from an early stage, becomes segregated from the rest in the future sexual organs of the adult, whence in conjunction with similar material from the opposite sex, it goes to form the succeeding generation in which it develops and differentiates, again, leaving a part to be continued on as before. Thus, as has been aptly said, "The parent is rather the trustee of the germ-plasm than the producer of the child, and with similar conditions to start with, and similar conditions in which to develop, therefore like tends to beget like." *

Injury or disease inflicted upon the soma, that is the part of the germ-plasm which by differentiation and development forms the body of the offspring, will not be transmitted, and as a large number of maladies produce no deeper effect than this, however serious they may be, it is claimed that few diseases are heritable. I need scarcely stay to point out that such affections as appear at or soon after birth, and are the result of infection or damage to the organism whilst developing, are in no proper sense of the word inherited, though they may be conveniently grouped as congenital. Should, however, the disease or injury have affected that portion of the germ-plasm set apart in the

individual for the continuance of the species,—resting as it were in the one generation to develop in the next ; then, by the nature of the case, the morbid condition will be transmitted, and hereditary disease occur ; and moreover, it is assumed that should the malady of the individual be of such a nature as profoundly to affect the general nutrition of the organism to such a degree as to involve the germ-plasm, then the state itself, or maybe some allied one, is propagated. I do not propose to do more than refer to the provisional hypothesis, put forward by Darwin, of pangenesis, and to point out that such a view is the exact opposite to that suggested by Weissmann, inasmuch as the ova and spermatozoa are regarded as constituted of gemmules derived from all the cells and all the tissues of the organism, including any part which has undergone any acquired modification, which thereby becomes transmitted along with the other and more specific structural characters of the individual to the resulting offspring ; and it suffices to say that whichever theory be adopted, the responsibility of the organ itself for the transmission of the morbid potentiality is obvious ; nor am I concerned to criticise Weissmann's theory, since it is only in its bearing on the part played by the individual in the transmission of normal or abnormal characters that I am at present interested.

If now we turn to consider what bodily states are to be regarded as heritable, we shall find that the number is very considerably less than was formerly supposed, and that the transmissibility even in those cases which are accepted, is not so potent as was thought. In other words, that it is less the disease itself than a predisposition or tendency towards it, often requiring for the development of the malady a suitable conjunction of external conditions, in the absence of which the individual escapes, though this cannot always be so, for the tendency may be so strong as to assert itself in spite of the most favourable environment.

Prominent among those conditions which tend to recur in successive generations are longevity and the reverse. Statistics have shown that the life duration is a particularly family characteristic, and reduced to its physiological terms this represents a fundamental quality of the living material of the body—bioplasm—together with the power of resistance or susceptibility to the effect of untoward surroundings.

* Article, "Heredity," by Prof. Arthur Thomson, 'Encyclopædia Medica.'

States which insensibly depart from the healthy type, reaching to a degree in which the well-being is interfered with, are represented by an undue formation of fat, obesity, or an exceptional leanness, and such states are notoriously prone to characterise the members of families over many generations.

A third group of morbid states which recur with sufficient frequency among relatives of successive generations comprise such essentially nutritive disturbances as diabetes and gout, and it is peculiarly noticeable that maladies of this class do not invariably "breed true," but one or other term of the series, in which is to be included obesity, may represent the underlying malnutrition.

Perhaps even more marked illustrations of disease appearing in the offspring, similar to those from which the parents suffer, are to be found among nervous maladies. The frequent transmission of such conditions is notorious. But a careful examination shows that whilst it is comparatively seldom that any special nervous affection such as chorea, tabes, or apoplexy occurs in members of the same family, what is far more commonly transmitted is some form of general nervous disturbance or undue susceptibility, such as neurasthenia, acting upon which unhealthy surroundings, *e. g.*, syphilis, alcoholism, mental fatigue, or anxiety, may determine some specific nervous disease.

Lastly, there are some affections, the nature of which are but imperfectly understood, such as hæmophilia and colour-blindness, which exhibit a remarkable liability to transmission, and curiously the former is chiefly manifested by males, whilst transmitted by females.

It is apart from my present purpose to attempt any exhaustive account of diseases that we consider as hereditarily propagated, from the frequency with which we observe their recurrence in successive generations, and it is probable that the list may even be shorter than it now is, as extended knowledge may show the absolutely essential requirement of some external factor for their manifestation. But so far as our present ideas go, I think I should find general agreement in assigning those I have mentioned to the class of hereditary diseases, leaving it an open question how far the predisposition is of itself sufficiently potent to determine the occurrence of the disease, or how far any environmental circumstance is requisite to develop it.

As further illustrating the responsibility of the organism itself in the occurrence and manifestation of disease may be mentioned the undoubted influence exerted by age and by sex. Even when all allowance is made for the varied external conditions attendant upon these states, there still remains a sufficient difference in the incidence of disease at different ages—that is the difference in susceptibility—and also in the manner in which morbid states manifest themselves. The far greater frequency of the acute infections in the young as compared to adult or old age, the diseases which characterise the periods of growth and those of decline, will serve to exemplify the one, as the far greater frequency in females of neuritis as a sequence of alcoholism, or of a mitral obstructive lesion in the same sex than in males, will illustrate the other.

A still more conclusive example of the responsibility of the organism is to be seen in the recovery from disease. A large number of the morbid states tend of themselves to revert to the normal; the disturbed metabolism swings slowly or quickly back to its healthy course, unless the damage it may have inflicted is too serious or profound. It is true that the completeness of recovery, or even recovery at all, may be hindered and retarded by unfavourable external conditions, but the potentiality of amendment is essentially intrinsic, and of this we take advantage when we adapt our therapeutic measures in such a way as to put our patients in the most satisfactory position for self cure.

Upon the intrinsic nature of the living matter itself, so far at least as the germplasm is affected (whether this be so inherently or subsequently by such changes in the soma as shall have reacted upon or involved it), must be dependent certain departures from the normal state, just as that normal state itself is primarily dependent upon the intimate molecular constitution of the bioplasm. But it must be admitted that it is more frequently a predisposition or tendency that is transmitted which may require for its development a favourable conjunction of the environment. The essential responsibility of the organism however remains undoubted. Equally so does this responsibility underly the principle of variation which determines the characters of the individual as opposed to the characters of the species, though this again may depend in some measure upon externals for its

exercise and for its perpetuation. Nor can the environment be credited with the production of those atavistic characters that represent latent or dormant ancestral qualities, nor the transmission to the offspring of the second male parent characteristics derived from the first, the phenomenon that is known as *telegony*, and upon the reality of which much doubt has recently been cast.* It must be the organism itself that is fundamentally responsible for their appearance, however some external factor may appear to have evoked them, and whilst these traits may without difficulty be conceived of as becoming manifest solely by intrinsic conditions of the organism, the environment alone can by no stretch of imagination be regarded as alone provocative of them.

Now it appears to me that just as we recognise that predispositions to disease rather than the disease itself is transmitted, a vulnerability of tissue that permits of an easier disturbance by external circumstances than normal, and that such predisposition or tendency is a property of the organism, so do we require some readjustment of our terminology, and though I am aware of the reluctance of some minds to pursue a precision in expression, I feel emboldened to advocate the use of certain terms, and the more so as they are not novel in themselves, nor originated by me, though I would strongly urge a consistency in their application.

In the word *constitution* we have ready to hand a word that may be taken to signify the sum total of the tendencies possessed by a living organism whencesoever derived, and whether beneficial or otherwise. But inasmuch as we must recognise that as there are variable forms of unhealthy constitutions which may be fitly termed *diatheses*, and even variable ways of being ill with the same malady, so there are several forms—I hesitate to say types—of healthy constitution, several ways, that is, of being well, for which the old word *temperament* seems properly applicable. And this, however much we may differ among ourselves as to the structural characteristics of these temperaments, or the influence they may exert in predisposing to disease. Essentially connected with these expressions is the idea of persistency, and

they should not be applied to any condition, whether of health or disease, which is expected to pass away and leave no trace.* With changing views as to the nature of disease and our extended knowledge, these words have undergone considerable alteration in the meaning attached to them, or they have been discarded altogether. Thus it is that "temperament" has come largely to denote certain mental characteristics which have some affinity with the physical conditions now referred to. Some there are who have refused to see any connection between different types of health and special proclivity to disease, and are not a little sceptical as to the reality of such types.† But it seems to me difficult to doubt their existence if hereditary predispositions are granted, though how far susceptibilities to disease are associated with the structural characteristics of normal temperaments precise observation alone would determine.

And here I would digress a moment to say how much I wish this Society could see its way to prosecute some collective inquiry of this kind, the ascertainment of clinical observations contributing to the settlement of undetermined questions. Other societies have done so with success, and the wide field of experience covered by members of the Medical Society of London should be sufficient if properly directed to furnish some valuable information, and I know of no subject that would be more interesting and worthy of such investigation than the incidence of disease in successive generations.

I have now shown, obviously with no pretence to originality, that whether the occurrence of disease be primarily determined *ab extra*, or its starting-point be essentially intrinsic, however much favoured by externals, the organism itself is in part responsible, and that that responsibility cannot be disregarded. In what I have said I have tacitly assumed the physico-chemical explanation of life which has been generally accepted for the past half-century, replacing as it did a vitalistic theory that in one or other of its many aspects had previously occupied the field for so long. At the present day, however, any one dealing, as I have imperfectly done, with the fundamental questions

* See Prof. Cossar Ewart's Address in the Section of Zoology, British Association for Advancement of Science, 1901.

* 'The Pedigree of Disease,' by Jonathan Hutchinson, F.R.C.S., F.R.S., 1884.

† "The Physiognomy of Disease," by Prof. Gairdner, in Finlayson's 'Manual of Clinical Medicine.'

which lie at the root of all biological knowledge is bound, if only for completeness, to make some reference to that recrudescence of vitalism in yet another aspect, which a small but distinguished body of biologists have recently fostered. Dissatisfied with the physico-chemical hypothesis as inadequate to explain vitality, though recognising the enormous benefits it has conferred in the prosecution of physiological inquiry, they have endeavoured to show not that the older vitalistic conceptions should be reinstated, since they also are far from satisfying though not without suggestion, but rather what has been termed a 'neovitalism.' This seeks to show that "the biological are separated from the physical sciences, not through the existence of any spectral line of demarcation between what is living and what is not living, but by the fact that the fundamental conceptions of biology are, and from the nature of the phenomena dealt with, must be, entirely different from those of physics and chemistry." The direction in which the explanation of these phenomena is to be found is in the pursuance of a method similar to that "which the morphologists have so successfully applied to the elucidation of structure." "The ground idea of the new anatomy was that of the existence of an immanent type, a plan which an organism or group of allied organisms adheres to through every variety of outward modifications. Adherence to morphological plan is evidently not the same thing as persistence of physical structure or configuration; for the physical configuration may vary enormously in cases where identity of morphological plan is perfectly clear." "The fundamental assumption of morphology is that each part of an organism is determined as regards its mode of existence by its relations to the other parts. . . . The results of embryological experiments illustrate very clearly the fact that the parts of an organism mutually determine one another's mode of existence. Now this implies a constant physiological determination of the nutrition of each part in accordance with the morphological plan of the whole. The morphological conception of an organism is thus just as much a physiological as an anatomical conception. As however the nutrition of an organism depends not merely on the relation of the parts to one another, but also on their relation to the physiological environment, it seems equally clear

that the conception in question implies that the influence of the environment also is determined in a similar manner to that of the parts of the organism. We are accustomed to look at environment simply in its physical and chemical aspects. It must be remembered, however, that what the environment is for an organism depends on the organism itself, and that the influence on the organism of any particular physical or chemical condition in the environment may vary indefinitely according to the nature or physiological state of the organism."

It is not my intention, even were I capable of so doing, either to support or to oppose these conceptions, and my purpose in making these quotations from the writings of one who is foremost in their advocacy is fully served by showing that if the effect of the environment on an organism is conditioned by the physiological state of that organism, then the responsibility for disease must be much greater on the part of the organism than I have ascribed to it. And I would add that some of the phenomena of disease appear to me strongly to support that view. It would certainly accord with the difference in result that follows the infliction of some detrimental external agency upon several healthy individuals similarly situated, and is consistent with the facts of immunity or of increased susceptibility, and also with the differences manifested during the course of the same disease attacking different persons.

I am aware how foreign to the general work of this Society is the subject that I have brought to your notice, and yet I feel ill-disposed to apologise for having taken advantage of my position on this occasion to treat of so theoretical a matter, however much I might do so for the triteness of my theme. For I hold that for the intelligent pursuance of our art, and for the clearer conception of the many problems it offers to us, we require some guiding principles, be they only hypotheses to be thrown aside as greater knowledge renders them untenable. I know how sternly matter-of-fact is the character of this Society, and how the results of observation or of practice meet here with a criticism that goes far to establish their results as facts of our science, and how strongly mere speculation is discouraged; yet knowing this I have dared to

* "Vitalism," by Dr. Haldane, F.R.S., 'Nineteenth Century,' September, 1898.

quote from a comment on the recent address of the distinguished President of the British Association for the Advancement of Science, who had made the atomic theory and its claim to continued support the subject matter of his remarks, I would say—"Facts, if they be well ascertained, are always worth seeking and possessing, yet little real progress can be made while they are merely accumulated and are considered separately or in small groups. Knowledge can only accrue when they are regarded in the relations in which they stand to one another and as perfectly accountable results of a fundamental law. This is so true that a wrong theory, as long as it enables phenomena to be considered in connection with one another, may be at times a more useful instrument than one which is accurate but more limited in scope."

Without giving full assent to the last statement, I am convinced of the wisdom of the general proposition, and I am a little inclined sometimes to think that in our steady avoidance of theory we sometimes, to our own detriment, miss a useful guide, and fail in taking a comprehensive view of our subject, so long as we hesitate to recognise the bearings and relations of the facts we so highly prize and strenuously seek after.

"*Observation*, however accurately performed, does not add to our *comprehension* of Nature, unless it is controlled by a scheme of thought for which confirmation or disproof is sought in the results of the observation. Some principle already shaped, whether it be a product of our own mental activity, or be handed on to us by authority, is wanted to give to each fresh item of perception a rank in an orderly plan, and to add clearness and value to that which would be, otherwise, unrelated and imperfect." *

But in endeavouring to form some estimate of the share taken by the individual in the determination and manifestation of disease, we are dealing with a subject that is essentially practical in its bearings, however speculative may be the basis which subtends the application. The correct estimation of the value of the facts ascertained when inquiring into the family history and life history of our patients, depends upon some knowledge of the workings of the organism in response to externals, and of such inherent qualities and

capabilities he may possess. Whilst it may seem but little towards the framing of a diagnosis in the use of that term which is restricted to the mere naming of the disease, in a wider sense of comprehending the real nature of the case it is all important, and underlying as it does prognosis, the estimate of the individual resistance to the disease, and furnishing thereon the only rational plan of treatment, its study cannot be too sedulously cultivated. I would claim therefore that I have not, after all, wandered far afield from our legitimate occupation, and whilst *dulce est desipere in loco* I venture to hope my dissipation in generalities has not been out of place.

Bad Effects of Camphor.—F. BOHLEN (*Deutsche medicinische Wochenschrift*, May 16th, 1901) reports two cases in which marked delirium followed moderate-sized medicinal doses of camphor. The first was a man with compensated heart disease, in whom the pulse was very small and thready, and a catarrh of the lungs was present. Three-fourths of a grain of powdered camphor was given every two hours. In the second case the patient was a woman who was suffering from heart symptoms following an attack of influenza. The same dose was given to her. After thirty-six hours the man had received nine and three quarter grains, and the woman nine grains. The effect on the hearts had been very satisfactory, but an intractable delirium had set in in both cases. At first this was not ascribed to the drug, which was continued. Bromine was given to quiet the condition, without success. After three days it occurred to Bohlen that the camphor might be responsible for the delirium; he therefore discontinued the powder and gave bromide alone, and was gratified by seeing the disagreeable symptoms disappear very shortly.—*Therapeutic Gazette*, September 15th.

MESSRS. BURROUGHS, WELLCOME & Co. have sent for examination some "tabloids" of morphine and emetine. The combination is a very serviceable one, and will be found useful in cases where a sedative and expectorant effect is desired for the alleviation of troublesome cough. The tabloid method of exhibiting a drug or combination of drugs lends itself to securing accuracy in dosage and purity in the quality of the ingredients.

* 'The Living Organism,' by A. Earl, M.A., 1898.

A CLINICAL LECTURE
ON
“ENTERIC FEVER.”

Delivered at Guy's Hospital, July 24th, 1901,
By J. W. WASHBOURN, M.D., F.R.C.P.

GENTLEMEN,—I will begin by reading to you the notes of a case of enteric fever recently in the clinical wards. The patient was a married woman æt. 28, who was admitted on July 11th for fever and pains in the abdomen. She came from a district where enteric fever was prevalent. There is nothing of particular importance in the family or personal history. She had been ill for nine days. On the first day of her illness she was suddenly taken with a shivering fit, and complained of very great pain in the head, which was so bad as to cause her to scream out. On the next day she sent for a doctor, who thought she had merely run down from overwork. The next day she again had to see the doctor, and she has been seen by him every day since, and finally, nine days after the attack had commenced, she was admitted here.

On admission she was in an apathetic condition and complained of slight pain in the abdomen. The tongue was red and slightly dry, and somewhat brown in the centre. The abdomen was rather fuller than normal. There were a few scattered spots on the abdomen and thorax. The spleen could be felt below the costal margin. The breathing was rather rapid, and a few rhonchi were audible on auscultation. There were no abnormal sounds in the heart. The pulse was 124, and the temperature was 105°.

On July 12th, the day after admission, the bowels were opened three times. The motions were liquid and of a yellow-ochre colour. On the 15th there is a note to the effect that the pulse was rather feeble last night, and it is the same this morning. The patient complains of very severe headache; the abdomen is not distended or tender. There is another spot on the thorax. On the 16th the note says the pulse is weak and rapid, and the patient was very restless early this morning. On the 17th there is again a note to the effect that the pulse is very feeble; the patient is no better. There is a slight trace of albumen in the urine. On the 18th the patient was much worse, the breathing of the Cheyne-

Stokes character, pulse very feeble, and abdomen distended; she died at 5 o'clock. The temperature during the time she was in hospital was about 103°. With regard to treatment, the diet consisted principally of milk, and for drugs she had chloral, strychnine, digitalis, and stimulants. The summary of the post-mortem examination states: “The right lung is oedematous. Two feet from the cæcum is a small ulcer perforating into the muscular coat. Below this there are numerous raised and infiltrated Peyer's patches; in a few ulceration has taken place, but in the majority there is none. The spleen is large, soft, and dark, and the rest of the organs are normal.”

This is a short account of a typical case of enteric fever, the main feature of which was cardiac failure.

She was attacked suddenly with a rigor and fever, but the disease was not definitely diagnosed until the ninth day, when she was sent into the hospital. At that time definite evidence of enteric fever had shown itself, there were spots, a slightly distended abdomen, an enlarged spleen, and diarrhoea, the motions being of the typical character met with in typhoid fever.

The principal feature of this case was the cardiac failure, and every day the notes laid special stress upon the weakness of the pulse.

In discussing some of the points arising from the above account, we will begin with the causation of enteric fever. It is a specific disease caused by a definite bacillus, which enters the body by means of the alimentary canal in the vast majority of cases, although exceptionally it may be inhaled.

The bacilli leave the body of patients who are suffering from the disease by means of the evacuations—the fæces and the urine. In the fæces the bacilli are always difficult to find; they never appear to be present in a large number, but in the urine they are sometimes present in very large numbers. You will have to consider the way in which the bacilli that are given off in the fæces and urine of a patient can gain access to the alimentary canal of a healthy individual, and so set up disease. An important point to remember is that a patient, sometimes for a very long time after convalescence, may continue to discharge bacilli by the evacuations, so that the danger arises not only from a patient who is actually ill with enteric fever, but also from a person some time after the fever has

completely subsided, and this is one of the great difficulties which we experience in dealing with the prevention of the disease.

Now you will readily see that people who are in attendance upon cases with enteric fever may contract the disease more or less directly. The hands may become soiled with the urine and fæces, and from the hands the bacilli may gain access to the alimentary canal. In this way nurses or orderlies who are in attendance upon a case of enteric fever sometimes contract the disease. This mode of infection can be avoided almost completely by cleanliness, and by the use of antiseptics for the hands immediately after attending to the patient.

The bacilli after having been passed out of the body of an infected patient remain living for some period, and they may multiply outside the body. Under suitable conditions various articles of food, such, for example, as milk, form good cultivating media for the growth of the bacilli; and if through lack of precautions, the food gets contaminated by the evacuations, enteric fever is liable to ensue. In some countries, such as South Africa, flies and dust may be the means of directly contaminating food with minute portions of the evacuations from patients suffering from enteric fever.

We do not know all the conditions under which multiplication of the bacilli will take place, but there is no doubt whatever that the bacilli under certain conditions may multiply not only in the soil but also in water. I remember once making some experiments with Mr. Pakes on the multiplication of the typhoid bacillus in water. We took some water and sterilised it in two ways. Part of it was sterilised by filtering through a Berkefield filter, and the rest by boiling. We introduced into both samples living typhoid bacilli, and we found that they multiplied much more freely in the water which had been sterilised by boiling than in that which had been sterilised by filtration. The explanation was this: In filtering the water we removed a good deal of organic matter that was present, by boiling we remove none; and the amount of organic matter present in the unboiled water was sufficient to form a nutrient medium for the bacilli. I am mentioning this experiment to show you what slight changes in the composition of the water may be sufficient to determine whether or not multiplication takes place.

We are still very ignorant of the exact conditions which favour multiplication of the bacilli in soil. All we know is that the chemical and bacterial composition of the soil, the temperature, and the amount of moisture are determining factors. The principal danger of contamination of the soil is a subsequent contamination of a water-supply derived from this source.

In England the contamination of water is the principal mode of conveyance of typhoid fever. Almost all large epidemics, such as, for instance, that at Maidstone, are due to the contamination of water, and in South Africa in most instances the large epidemics among the troops have been caused by water. You must, however, remember that water is not the only mode of conveyance of enteric fever, and that in an epidemic several modes co-operate in forming the epidemic. For instance at Maidstone, after the water had been shut off and a pure supply had been provided, cases still continued to crop up. These cases occurred in the houses, or the neighbourhood of the houses, of patients suffering from the disease, and arose no doubt from more or less direct contamination of articles of food.

The prevention of enteric fever consists in measures to avoid contamination of water and food with typhoid bacilli. The medical man in charge of a case of enteric fever should see that the urine and fæces are disinfected, and that great care is taken in the disinfection of the linen. The nurses in attendance should be instructed to disinfect their hands frequently. As the urine often contains a large number of bacilli it has been suggested that urotropine should be administered to the patient in doses of ten grains three times a day in order to render the urine sterile. Unfortunately there is a certain amount of danger attached to its employment. I have known of two instances in which severe hæmaturia has been set up by the use of urotropine.

The careful disinfection of the excreta of patients suffering from enteric fever will do a great deal to prevent the spread of the disease, but it is not sufficient to stamp out the disease for two reasons—the bacilli are discharged in the excreta during the early stages before the disease is recognised, and they continue to be discharged for some time after convalescence, when disinfection is not possible. The only way by which we can hope to

stamp out enteric fever is to adopt such sanitary measures in the disposal of human excreta as to preclude the contamination of drinking water, either directly or indirectly, through the soil

Having spoken thus to you about the causation of enteric fever, we come to consider the course of the disease. After the patient has ingested the bacillus of typhoid fever a period of about fourteen days occurs, during which, as a rule, nothing particular is noticed. This is the period of incubation. It is very interesting to speculate on what takes place during these days of incubation. There is no doubt that during these fourteen days some important changes must be taking place in the body, but as to its nature we know very little. In South Africa, where I have had many opportunities of watching cases of typhoid fever from the very beginning amongst nurses, orderlies, and medical officers, I have often observed short febrile attacks to occur during the incubation period. One is called to see a patient, and finds that he or she is suffering from slight fever, with a temperature of 101° or 102° ; this goes on for two or three days without other symptoms than a general feeling of malaise, then the temperature goes down, and you imagine the patient is well, and he goes back to duty; but in a few days he is seized with a typical attack of enteric fever.

The onset of enteric fever is usually gradual. The temperature sometimes rises in a staircase fashion, each morning temperature being a little higher than the morning temperature of the day before, and the evening temperature a little higher than the evening temperature of the day before, but each morning temperature is lower than the evening temperature of the preceding day. The maximum is reached in three or four days. Sometimes the temperature rises in an irregular fashion; it may be normal in the morning, and rise to 102° or 103° in the evening for several days, and then the morning temperature rises. Sometimes the temperature rises suddenly with a rigor like an attack of typical pneumonia, as appears to have been the case with our patient, who was suddenly taken ill with a shivering fit. During the period of onset, which lasts several days, a number of symptoms may arise; one of the most prominent and characteristic is headache, which, as a rule, starts in the frontal region. In South Africa a patient with bad headache and with

fever was almost sure to be going in for typhoid fever. In the case I have quoted headache was a prominent symptom. Besides headache there may be pains in various portions of the body; pain in the back is by no means an uncommon symptom, so, too, is pain in the neck. I remember several cases in which the patient complained of such severe pain in the neck that we thought there must be some local inflammation. Another common symptom is pain in the back of the eye, just as occurs in influenza.

As a rule the pulse is, during the onset, more rapid than it is at a later period. Insomnia is often a marked symptom. The patient will say: "If I could only get some sleep I should be all right." The patient is generally restless, and he may suffer from various neurotic disturbances. Sometimes the disease starts with vomiting, which continues for a day or two. Not infrequently epistaxis is one of the commencing symptoms; in England I think it is very common. The patient sometimes complains of his throat, and the tonsils and soft palate are found to be a little swollen and reddened.

During this period of onset a diagnosis is quite impossible, but after several days things will begin to settle down. The temperature reaches a more or less constant level of about 103° , with evening rises and morning falls. At the same time the various symptoms above described—the pains, the headache, and the insomnia—begin to disappear, and the patient becomes drowsy and apathetic. This is the stage in which you will generally see the patient in hospital practice. You rarely see the case in the early stages, and that is why I have laid some stress on the symptoms which occur during that period. By the end of the first week or the beginning of the second week, certain characteristic symptoms appear. One of these is an enlargement of the spleen, which can be best ascertained by palpation. The best method is to stand on the left hand side of the patient, and on a level with his head; the tips of the fingers are placed just below the costal margin, and the patient is told to take a deep breath. The spleen can be felt as it descends.

The next symptom is the eruption, which consists of small, slightly raised spots, which you can feel with the finger, and which disappear on pressure. They are of a rose colour and are situated chiefly over the abdomen, but are often found on

the back, and not infrequently in other parts of the body—on the extremities, the chest, and even on the face.

The condition of the bowels is often characteristic, there is diarrhoea and the motions are of a light yellow colour. There is not always diarrhoea, there may be constipation all through. In South Africa constipation was the rule, while in England diarrhoea is the rule. The tongue is usually covered with a thick brown fur, except at the tip and edges, but sometimes you will find the tongue clean all through the attack, not only in mild cases but also in fatal cases. The abdomen is almost invariably slightly distended, but it may have a normal appearance all through the attack.

Another common symptom is deafness, which appears to be of central origin, and which disappears as the disease subsides. Slight bronchitis, too, is a common symptom. Delirium, generally of a quiet character, is frequent.

Such are the symptoms of the second stage. They begin about the end of the first week and they go on till the beginning of the second week. Day by day there is very little change in the patient; and then, about the beginning or towards the middle of the third week, the patient improves and a change in the character of the temperature occurs. The temperature may come down gradually in the same staircase manner in which it rose, or what is much more common, it becomes irregular, the morning temperature being normal and the evening temperature still raised. Towards the end of the third week or the beginning of the fourth the evening temperature no longer rises.

Now the above is a description of the usual course of an attack of enteric fever. First you have the incubation period during which you may get some febrile disturbance, then the period of the onset of the disease which lasts for several days, during which the temperature is irregular, and the patient is restless and suffers from various symptoms, such as headache, pains in different parts of the body, insomnia, neurotic disturbances, and so on. Then comes the second stage, during which the temperature remains at a high level, with only slight morning and evening variations; during this stage the previous symptoms subside, and the patient becomes listless and apathetic, and various characteristic symptoms arise, such as

enlargement of the spleen, rash, and distension of the abdomen. Then comes the third stage, beginning some time in the third week of the disease, in which the patient begins to get better, the first sign being a change in the character of the temperature, which sometimes in a regular staircase fashion, but more often irregularly, comes slowly down to normal.

This is the usual course of an attack of enteric fever, but recovery by no means always ensues. The temperature, instead of falling during the third week, may remain high, and the patient may die of exhaustion, or death may occur as the result of some complication, such as perforation or hæmorrhage.

There are several types of enteric fever. There is what we may call the *toxic* type. The patient does not suffer from complications, but he appears to be severely affected with the virus of the disease, and, as a rule, the symptoms are severe from the beginning. The patient is quite overcome with the disease—he is collapsed, the breathing and the pulse are rapid, and there is great muscular prostration. These symptoms are accompanied by high temperature, and the patient dies during the second week of the disease.

In the *cardiac* type, under which heading I place the case we have described, the main brunt of the disease falls upon the heart, which is affected, as a rule, from the beginning. The pulse is rapid and feeble, but the patient is not otherwise severely attacked. There is not, as a rule, delirium or insomnia, there is no evidence of severe affection of the nervous system, and the abdominal symptoms are not marked; but the heart is severely affected, and these patients often die quite early, twelve or fourteen days after the disease has commenced. You often meet with this type of enteric fever in patients whom, except for the condition of the heart, you would consider to be suffering from a mild attack of the disease. I have noticed in South Africa that the patients who are subject to this type of the disease are those who have been exposed to great privation and fatigue. The heart which has been overworked is the first organ to suffer from the poison of the disease.

There is another type which you may call the *abdominal* type, in which there is severe affection of the Peyer's patches, and deep ulceration. The

tongue becomes very foul, and its surface dry and cracked, and sordes collect on the lips; the abdomen is very distended; there is considerable diarrhoea; and as a rule a high and more or less irregular temperature; there is much delirium, which passes into coma. Now this type does not manifest itself at the beginning of the case. It is only after the disease has gone on to the end of the second week or the beginning of the third week that these symptoms appear. No doubt they are due to deep ulceration and sloughing processes occurring in the intestines, caused by secondary invasion of the tissues with other bacteria.

Then there is another type, and that is the type of *prolonged fever*. At the end of three weeks, instead of the temperature becoming normal it continues raised for another three weeks. As a rule there is some indication of the temperature coming down at the end of the first three weeks, and it would appear that these are cases of relapses arising before the primary fever has subsided.

There is another type, the *mild* type, comprising cases with the usual symptoms—enlargement of the spleen, fever, and so on, the only difference to the ordinary attack being that the symptoms are mild. There are other mild cases with no very definite set of symptoms at all; the only symptom you can be sure of is the fever. There are no spots, no enlargement of the spleen, no diarrhoea, but a temperature lasting for a period of two or three weeks. In other cases the temperature only lasts a few days, and the case is only recognised by the occurrence of a relapse. I have in my mind a case of a patient who had a fever lasting exactly a week. His temperature went up to 103° , and gradually came down, and at the end of the week it was normal. There were no symptoms whatever, no enlargement of the spleen, no swelling of the abdomen, no loss of appetite. At the end of a fortnight he had a relapse, and during that relapse the ordinary symptoms of enteric fever appeared.

Before concluding I would just like to say one word about relapses and the second attack. Relapses not infrequently occur, and they are of the same nature as the primary attacks; as a rule they are mild, but sometimes severe and even fatal. A relapse comes on, as a rule, in about a fortnight

after the fever has subsided. It may occur as late as three weeks, or even later, but at the end of three weeks you can for all practical purposes say that the patient is free from the danger of a relapse. One attack of enteric fever does protect against a second attack to a very great extent, but it does not protect absolutely. In England a person rarely gets a second attack of enteric fever, but in South Africa second attacks are not uncommon. The reason appears to be that exposure to infection is greater in South Africa than in England.

Now inoculation against typhoid fever consists in injecting the dead products of the bacilli into the body. The theory is that you produce what corresponds to a mild attack of the disease, and the idea is that this mild attack will protect against another attack. With regard to the practical results of inoculation, my own experience is that it is of very little value. I am quite satisfied that it does not modify the course of the disease. I have not observed any material difference in the nature of the attack between the inoculated and the non-inoculated. The fact that enteric fever does not protect to the extent we previously thought will explain the unsatisfactory results.

There are many other points, gentlemen, which I should have liked to discuss, but time compels me conclude.

Treatment of Conjunctivitis.—M. F. Terrien divides his subject into two groups, the treatment of specific and of non-specific conjunctivitis. He is opposed to bandaging the eyes for catarrhal conjunctivitis. When secretion is abundant, a solution of 1 or 2 per cent. zinc sulphate may be used as a collyrium, to be used three or four times daily. Cocaine is contra-indicated on account of its tendency to cause desquamation of the cornea with possible infection. Protargol in a solution of one fifth of one per cent. is also a good collyrium. As a prophylactic against blennorrhagic conjunctivitis, the author advises irrigation of the mother's vagina before the birth with a 1 to 5000 bichloride of mercury solution, and Cr  de's instillation of nitrate of silver. If the disease develops, cauterization of the lids with nitrate of silver solution (2 or 3 per cent.), followed by irrigation with a normal salt solution. Protargol should also be used. Canthoplasty may have to be performed. Frequent irrigations and ice compresses also form part of the treatment.—*New York Med. Journ.*, September 21st.

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A

PLEA FOR CÆSAREAN SECTION.

A Lecture delivered at the Medical Graduates' College and Polyclinic, June 26th, 1901.

By Professor W. JAPP SINCLAIR, M.D.

GENTLEMEN,—I wish to address to you a few words this afternoon upon the subject of Cæsarean section—an argument and an appeal with a practical object, founded on the history of the operation. In all the long story of human suffering no surgical operation has so completely laid hold of the imagination and excited such profound and sustained professional interest as Cæsarean section. Its history takes us back to the region of fable and tradition. In the middle distance the history presents itself to us as a hazy mass of facts and opinions, which is neither exact professional knowledge nor altogether fable. It is only in comparatively recent times that we meet with clearly chronicled histories of cases, so exact and detailed that we can employ them for purposes of generalisation and practical guidance almost as we do those which are produced in our own time. But if much of the history of the operation is unsatisfactory from a professional point of view, it is always fraught with the deepest human interest. There is almost always the spectacle of two human lives at stake with the maximum of human suffering; a woman existing in agony for hours, or days, or even in some rare recorded cases, for weeks, in the throes of parturition. But not only is there the spectacle of physical suffering; it requires very little imagination to picture to ourselves the mental torture of the sufferers, and the prolonged agonising distress of those interested in their fate. The crisis in each case is reached by the sufferer's submission to the necessary torture of an operation, and then the painful episode is almost invariably brought to a close by death.

To the professional reader the history of Cæsarean section is of unflagging interest. The

aversion to the operation produced by its almost invariably fatal result led to the evolution of all sorts of false theories on the necessarily mortal nature of certain wounds, for instance those of the serous covering of the uterus, and of the equally fatal termination of the disease then called "malacosteon." We see also in the story the dawn of abdominal surgery. We mark the earliest rudimentary forms of some of those methods which in the later years of the nineteenth century led to the greatest success in abdominal operations, as, for example, flushing of the abdomen and drainage, which are clearly enunciated by the Cæsarean section operators of the time of the French Revolution. The medical history of the nineteenth century offers grounds for congratulation on the results which have been achieved; but there is also very much to be regretted regarding the level of knowledge exhibited in the general routine practice of midwifery. There is much that might be called sinning against the clearest light, for it is not so much want of knowledge of what can be done which perpetuates the practices that are most to be regretted; it is the absence of a clear apprehension of the attainable amount of success, and the consequent want of that faith and confidence which lead to prompt and decisive action. If we were all fully impressed with the lessons which the history of Cæsarean section teaches, we would shake off the apathy which affects our judgment regarding alternative proceedings, and some of these would soon shrink into insignificance, or be banished from our obstetric practice as discreditable.

It is not necessary to go back to the ancient history of the operation for the facts upon which the argument is founded that I wish to bring before you. For this purpose the last few years of the nineteenth century are almost sufficient. It is chiefly from the recorded experience since the introduction of antiseptics into abdominal surgery, and the adoption of certain modifications of the Cæsarean section, that we have to draw the inferences that must guide us in our practice. Nevertheless it would be interesting and instructive to go back to the year 1750, the point from which Baudelocque started in his literary researches and inquiries, and review the period during which he and his contemporaries and immediate predecessors were engaged in the active work of their

profession, and to note, as it were, in each decade the changing opinions of the obstetric surgeons, until we reach the point of view which is made possible by the introduction of antiseptics and of modern methods.

Baudelocque was called upon to defend the Cæsarean section before the Society of Medicine of Paris, to support *inter alia* the thesis that the Cæsarean operation is not inevitably fatal to the mother. "Citizen Baudelocque" obtained a formal vote from the Society "that this operation, however dangerous it may be, has been often practised with complete success." He had proved his thesis. This was in September, 1798. At the present day I do not think that if we were always to act according to our available knowledge in dealing with the cases which present absolute indications for the Cæsarean section, we should take up a too extravagantly optimistic position if we were to maintain the thesis that there is no inherent mortality in Cæsarean section, and that no mother should die from the operation. But between the time of Baudelocque and the results obtained by the conservative Cæsarean operation at the close of the nineteenth century many gloomy pages of the history of obstetrics have had to be written. Pajot, the predecessor of Tarnier who has only recently died, said of Cæsarean section, "This operation has cost the lives of all the unhappy women who have undergone it in Paris since the beginning of this century, and still they go on operating." This statement was not perhaps literally true and exact, but it indicates the state of professional opinion. It might have been the utterance of Ambrose Paré or Mauriceau a century or two before, or of our own Sir Fielding Ould, almost the only English writer of those times who mentions the operation, and who denounced it as "this detestable, barbarous, illegal piece of inhumanity." Similarly picturesque language was used by Simmons of Manchester in his controversy with Hull in the first year of the nineteenth century. The state of feeling with regard to Cæsarean section towards the end of the eighteenth century is very well illustrated by the excitement produced by the first successful symphyseotomy. There is no more remarkable episode in the history of obstetrics. In 1777 Sigault, of Paris, a young French surgeon, performed the operation for the first time, and the patient survived. The child was also saved. The

story was immediately noised abroad all over Europe, and in every great city there was some obstetrician ready and willing to perform the operation. The subjects for the operation were readily found. There was a strange excitement, not mere interest; the Academy voted a pension, they struck a medal, and did everything in proportion for this happy surgeon that we do now for a great and successful general after a campaign. Jan Petersen Michell, of Amsterdam, in his 'De Synchondrotomia Pubis Commentarius,' published in 1783, gives a prodigious list of pamphlets and articles which had already been published on the subject, and he mentions twenty-five cases of the operation. These incidents are best interpreted as affording a sort of measure of the aversion with which the Cæsarean section was then regarded by the public and the medical profession. It was a demonstration of welcome to an operation which promised to be a comparatively safe substitute for its dreaded alternative.

Mauriceau's position gives another illustration of the professional aversion to Cæsarean section. He was the great authority of his generation, which was about half a century before the time of Baudelocque. Mauriceau went the length of maintaining that no proceeding was justifiable in certain conditions said by some to indicate the Cæsarean operation, except to allow the woman in labour to die, and to be ready to perform Cæsarean section just as she was *in articulo mortis*, in order to save the life of the child.

A reaction soon set in with regard to symphysectomy. Baudelocque showed that the mortality resulting from it was extremely high. As usually happens, and as is too well illustrated in novel proceedings introduced in our own time, the operation was abused; that is to say, that owing to lack of judgment it was applied to unsuitable cases, and the general result was a mortality of 25 to 40 per cent. or even higher. Baudelocque said, "the new operation was practised more frequently at this period (1785) in the space of seven years and a half, than the Cæsarean section in a quarter of a century, and the general opinion began to change." The operation really soon became obsolete; then it fell into oblivion, to be resuscitated unfortunately time after time, and especially in recent years in Paris by Pinard, and in Naples by Morisani.

It is nowadays usual to represent the results of the Cæsarean operation in the eighteenth century as worse than they really were, in order to make the contrast more striking when the object is to call attention to the results of the last decade of the nineteenth century. Baudelocque collected seventy-three cases, with forty-two deaths; but justly deducting 11 moribund patients as victims of "causes foreign to the operation," he proved the result to have been a mortality of 50 per cent. In England no case of genuine Cæsarean section had been successful up to the end of the eighteenth century.

From this time there is very little to record with regard to the practice of Cæsarean section for fifty years or more. Men's minds turned once more from symphysectomy and other alternatives to Cæsarean section in cases of extreme difficulty, and the operation had to be occasionally performed, but always as a last resort. There was a certain amount of relief afforded by the introduction into this country of what was a new method of treatment in contracted pelvis, namely, the induction of premature labour, chiefly owing to the influence of Denman, at the beginning of the nineteenth century. But all through the first three quarters of the century there was practically no improvement in the mode of operating or in the results obtained. Occasionally there was a successful case; but though it was rarely practised, the operation was much discussed. Our Blundell, writing in 1828, says that he sometimes thought that Cæsarean section might be more successful if the uterus were removed altogether in completing the operation. Dr. Thomas Radford, of Manchester, who was the most successful operator of his generation, had two successful cases out of five, and his are the only successful cases which I have been able to find recorded up to that time in England, with the exception of a doubtful case at Blackburn. Radford wrote in 1849 in very hopeful terms as to the future of Cæsarean section. He said, "I am justified in advocating it as an operation of election, not merely having recourse to it as one of necessity, according to our present obstetric rule, when no other means can suffice."

The introduction of anæsthesia wrought no change in the results of this operation, for there was one fatal defect in the surgical proceedings. There is nothing instructive in the history, and

nothing to argue from, until the introduction of antiseptics. If you look into the English midwifery books of the time between 1870 and 1880, and even considerably later, you will find there had been no improvement in the position of the Cæsarean section as a surgical operation from time immemorial. The mortality is stated to be 95 to 100 per cent. The operation lies under the shadow of a gloomy tradition; it is still looked upon as a last resort, and nowhere is there to be found an original idea concerning it, or a hint that improvement is possible. Take Leishman, for instance, in the edition of his 'System of Midwifery' published several years after Porro's first operation; referring to the Cæsarean section, he says, "The maternal mortality in this country has been so great—not less than 85 per cent. of all recorded cases—that a very general idea prevails that this is almost exclusively a child's operation." Although he feebly combats this conclusion, he sets down as his own opinion "that we are driven to this last resource whenever we recognise the fact that the foetus, however mutilated, cannot be extracted by the pelvic canal." Meadows, whose manual was in general use among medical students a quarter of a century ago, touching on the crux of the question, states, "As a rule I should certainly object to this foreign substance (the sutures) being introduced, and I would rather trust to uterine contraction for securing apposition of the cut surface." Barnes even later says something to the same effect; and there is a curious passage in Galabin's manual, published in 1886—that is to say four years after Säger's famous proposal appeared in the 'Archiv für Gynäkologie.' He says, "The great source of danger in Cæsarean section does not appear to be capable of removal by any modern improvements in abdominal surgery. It lies in the fact that through the wound in the uterus there is a path for septic matter to reach the peritoneal cavity . . . and that immediate closure and primary union of the uterine wound cannot be obtained with certainty." No clearer statement could be made calling attention to the chief cause of failure of the operation since it was first performed in the dim and distant past. Only one step forward—the proposal to close the uterine wound—and the operation might have been placed much sooner in its present comparatively secure and happy position.

My object in giving these quotations from the writings of men of light and leading amongst us—and the extracts might be multiplied indefinitely—is to show that we have all along been weighed down by tradition; the phenomenon of free and original thought has only glimmered out now and again during the course of a century, and in spite of foreign inspiration we are not yet emancipated. It is humiliating to read in one of the most recent English manuals the constant recommendation of craniotomy on the living foetus, and even of the repeated procuring of abortion in cases of contracted pelvis. In this respect, at least, the work is a century behind Denman professionally, as it is chronologically. It is, however, an excellent exponent of the "conservatism" of English obstetrics.

During this long period of stagnation of thought and action in England, considerable progress had been made on the Continent, especially in Germany. Michaelis and E. Martin operated successfully, the latter suturing the uterine wound to the abdominal wall. Still the chief cause of the mortality of over 50 per cent. was sepsis of the uterus and septic peritonitis; it accounted for at least half of the total number of deaths. Then came a sudden revolution in the position of the operation. Two of the most important modifications ever proposed came in rapid succession. Porro, of Milan, observing that the fatal sepsis appeared to spread from the uterus, asked himself, "Why not remove the uterus?" That was the operation. Removing the uterus immediately after extracting the child was Porro's operation. It was first performed in May, 1876. Many cases were soon reported from all over Europe with the exception of England, and the mortality sank to one half its former proportion.

Still it remained high until a few years ago. Taking the last statistics by Harris, giving the results from 1876 to 1891, we find that the operations, as far as he could learn, numbered 441. Of the patients 274 died, a mortality of 61 per cent. But the operations were the work of 224 surgeons, and 154 of them performed only one operation. The result of 150 cases in the hands of men experienced in abdominal surgery, showed a mortality of forty-three, equal to 28.5 per cent. From these figures the most despondent might well take courage, and confidently anticipate still better results.

But how were the better results to be obtained? Considering that the appalling mortality of the old Cæsarean operation was attributable chiefly to sepsis, and that the sepsis arose from the escape of fluid through the wound in the uterus, and that the mortality after amputation of the uterus was still as high as abdominal hysterectomy for fibromyoma, Säger proposed exact suturing of the uterine wound. That is Säger's operation—'Der Conservative Kaiserschnitt.' It was the adoption of this organic detail, the crowning-point in the technique of the operation, which led to the brilliant successes of the remaining part of the century. Very soon from nearly all the lying-in hospitals of the civilised world came reports of successful operations. The most striking results were obtained in those countries of Europe where osteomalacia is a not uncommon disease, where so often during labour the pelvis is found deformed to the extent of making even craniotomy difficult and dangerous, or altogether impossible. This disease, which at the beginning of the century was supposed to render inevitable a fatal termination to Cæsarean section, was found at the end of the century to be cured or arrested in its course by Porro's operation.

Since Säger's method of operating came to be generally adopted the results have steadily improved. Some minor details have been omitted, and many have been introduced by experienced operators. There has been much controversy about the value of some of these details, but it may be confidently alleged that their influence upon the result in the individual case is comparatively trifling if only absolute asepsis is secured. To this end the decision to operate must be promptly arrived at before the patient has become exhausted by prolonged and unavailing labour, or before she has been subjected to futile and injurious attempts at delivery, as used to be the practice in the early part of the nineteenth century—and even later.

I have said that the mortality from the operation rapidly diminished, and consequently the number of operations rapidly increased. Men began to consider not merely absolute but also relative indications in certain categories of cases. Now, instead of 60 per cent., to say nothing of the 80 or 90 per cent. mortality, as stated by some of our English authorities in comparatively recent manuals, I find that taking the cases all over the world up to

the end of the nineteenth century the mortality comes out at 13 per cent. in thoroughly authenticated and clearly recorded cases. For the Porro operation the percentage comes out at 14·8 per cent., so there is very little difference. From those figures, which are the result of a long and tedious investigation of available writings, comfort may be derived. I wish further to lead up to certain facts from which important practical inferences may be drawn, namely to the results obtained by experienced operators when they were dealing only with cases which were unquestionably suitable for operation.

We have to deduct a large number of cases which are brought to the hospitals in a moribund or desperate condition, in which efforts have been made to deliver by forceps, by version, or even by craniotomy; where labour is complicated by obstructing tumours; cases in which the operation has been done for eclampsia; and cases of advanced cancer, where the patients have died afterwards of septic peritonitis from bacterial invasion. Now deducting all these, whilst investigating the work of experienced operators only, I have compiled a list from the Continental journals and other sources, partly from correspondence, and the mortality comes out at 4·5 per cent. on over 400 cases remaining after the process of elimination. The result for the Porro operation—which I believe to be more dangerous than the conservative operation in suitable cases—obtained in certain German clinics for ten years, viz. those of Chrobak, Schauta, Leopold, and G. Braun, shows a mortality of 2·5 per cent. It must be admitted that the Italian and the American results compare very unfavourably with those of the selected German clinics.

Now, conceding every reasonable demand that may be made owing to the weaknesses of the statistical method, it must be admitted that the recent results of the operations are surprisingly favourable. They show what success is attainable, and suggest efforts to eliminate the remaining factors of failure. We naturally ask ourselves the question—How have these results been obtained, and what more can be done? Keeping always in mind the exact suture of the uterus in the conservative operation, the most important results have been obtained on the Continent by distinguishing between antisepsis and asepsis. There has too frequently been a certain amount of carelessness in the surgical work, a trusting to antiseptics instead

of keeping things absolutely clean. From success also there has arisen a greater hopefulness, and consequently operation has been resorted to earlier. Added to this there has been improved technique, and the operators have become more experienced and more skilful and efficient. Why then should there be any mortality at all from the operation? One cause of the still existing mortality is, I believe, a certain faultiness remaining in the technique; but especially, and by far the most important factor in producing a fatal issue is injudicious interference previous to the operation, and delay owing to the want of a fixed resolution to adopt and pursue a definite and distinct line of action. Let us consider all these factors together.

1. *What is the best time for operating?* I would urge that in the class of known deformity to which I wish to call your special attention, the operation should be done before labour begins. The notion that the uterus will not contract when it is prematurely emptied, or that Nature is not prepared for the processes of normal involution, is just as well and no better founded than the ancient conviction that an injury to the serous covering of the uterus was a mortal wound. It is an opinion contrary to experience. The everyday observation of the induction of premature labour, or emptying the pregnant uterus when the patient's life is in jeopardy, and what is more to the point, a long array of successful operations performed before the onset of labour, prove the objection to be groundless. The operation should be done at a time to suit everybody concerned; it should be arranged for as is now usual in cases of ovariectomy, or of operation for tumour of the uterus. When labour has set in the least time possible should be lost. v. Winckel considers the best time to be the middle or end of the first stage; but he does not support his opinion by any convincing argument drawn from experience. My own experience of operating on primiparæ enables me also to say with confidence that drainage through the cervix may be perfect, even if no labour pain has occurred.

2. *Which is the best anæsthetic for Cæsarean section?* This may seem a trivial question, but it seems to me to have a certain amount of importance. Out of my fourteen cases two were in great danger from ether bronchitis, and others suffered more or less. I consider ether absolutely unsuited for Cæsarean section cases, and I shall

never have it administered again, if I can help it, to a patient on whom I have to operate. I much prefer chloroform. Some of our patients suffer from "winter cough," and in ether administration there is then a special danger of setting up acute bronchitis. But it is possible that the place, even of chloroform, may be taken by subarachnoid injection of cocaine. In my last case of Cæsarean section, as yet unpublished, the patient, an elderly primipara, suffered from chronic bronchitis, dilated heart, and albuminuria, and was, I am afraid, an alcoholic. I did the operation after a special cocaine solution had been injected into the patient's lumbar region, in the way with which you are all no doubt familiar. The result was very striking. It took only five minutes for the patient to become anæsthetised from the waist downwards. She was then blindfolded, and rendered deaf by cotton wool inserted into her ears, so that she should not see or hear anything to shock her, and a nurse stood by her to comfort her during the progress of the operation. She was at first a little sick, but she complained of no pain, and simply asked once or twice for a drink. The use of this particular method of anæsthetising was not intentionally an experiment. The administration of a general anæsthetic seemed too risky, and I had no confidence that the local use of cocaine injected into the abdominal tissues, as occasionally practised in Germany, would prevent suffering amounting to torture. Barring some peculiar nervous symptoms the patient did well, and made one of the best recoveries I have seen.

3. *The abdominal incision: its position and dimensions.* There is no controversy nowadays about the site of the wound in the abdomen, but towards the end of the eighteenth century there was a chronic discussion as to which was the best place for the incision, and who was the originator of the wound in the linea alba. Each operator had a fancy cut of his own. Rousset, who was the first operator about whom we have any definite information in this respect, operated by an incision outside the rectus muscle on either side. Levret made an oblique cut. Lauerjat incised transversely; Stein made also an oblique incision, and Baudelocque was the first to make the wound in the middle line. The wound should obviously be a sufficiently large one; it should be made as large as may be required to admit of prompt action in manipulating and incising the uterus. The centre of the wound will be found, as a rule, to be near the umbilicus. For the Porro operation it should be considerably lower down.

(To be concluded.)

A CLINICAL LECTURE ON THE NASAL DISCHARGES OF CHILDREN.

Delivered at the Hospital for Sick Children, Great
Ormond Street, June 27th, 1901,

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LADIES AND GENTLEMEN,—The subject which I have chosen for to-day is perhaps unfortunate in one way, namely that I cannot illustrate it by living cases; it is however so important that it is well worth considering in spite of this, for I am sure there is often considerable difficulty arising in the diagnosis of certain cases of nasal discharge in children. I propose, therefore, first of all to give you a list of the most important causes of nasal discharge in children, and then to go over the chief points in connection with the diagnosis in a given case, and after that, if there is time, to indicate some of the most important points in connection with each condition.

Most of the important causes are included in this list, which, however, must not be considered to be complete. The causes are arranged as much as possible in the order of the frequency in which they occur. I have divided them into two classes, because I think it is an important subdivision—first, those which produce a bilateral discharge involving the whole of the nasal cavities; and secondly, the causes of a unilateral discharge.

Causes of Nasal Discharge.

I. Bilateral :

- (1) Simple catarrh, which is not of any very great importance, but is very common.
- (2) Rhinitis associated with adenoids.
- (3) Congenital syphilis.
- (4) Diphtheria.
- (5) Infection at birth.
- (6) Atrophic rhinitis.
- (7) Tubercular disease.
- (8) Other rare conditions, including such things as malignant disease in the nose and maggots in the nose, which are very rare in England but are seen oftener abroad.

II. Unilateral Discharges :

- (1) Foreign bodies.
- (2) Unilateral nasal diphtheria.
- (3) Sinus disease, including the antrum, frontal sinus, and occasionally the ethmoid cells.
- (4) Tubercular disease.

So much for the causes. I propose next to go into the question of diagnosis in any given case. Suppose a child is brought suffering from a discharge from the nose, let us think of the chief points which would guide us in making a diagnosis. We will consider these under the following heads :

- (a) Age.
- (b) History.
- (c) Duration.
- (d) Character of the discharge.
- (e) Is it bilateral or unilateral?
- (f) Physical examination.
- (g) Other symptoms present.
- (h) Bacteriological examination.

With regard first to *age*, in the nasal discharges of children this is not of much help, but if the infant be a very young one, say within a fortnight or a month of birth, it is nearly always a fact that the discharge is due to one of two things: either *congenital syphilis*, so-called snuffles, or it is due to No. 5 in the list, *infection at birth*, which is the same thing as blennorrhœa neonatorum, but affecting the nasal mucous membrane instead of the conjunctiva. That is the only help that age will give us in arriving at a diagnosis.

Secondly, there is the question of *history*. On running through this list you will see that the previous history may be of a certain amount of assistance in some cases. For instance, in *congenital syphilis* you may get a history of other children of the family being affected with various syphilitic lesions; or you may get a history of repeated miscarriages, or of children being born dead. In the case of *diphtheria* there may be a history of other cases of the disease in the same house occurring at the same time, particularly faucial diphtheria. Or you may find that children have been removed to a fever hospital suffering from diphtheria. As to *atrophic rhinitis*, there is evidence that this is to some extent hereditary. Instances have been described in which several

cases have occurred in the same family, so history may help you also in this connection.

With regard to *tubercle*, although it is not by any means constant for tuberculous disease in the nose to occur in those in whom there is a tuberculous family history, this certainly is so in a large proportion of the cases.

If a *foreign body* is in the nose, history may again assist. I remember very well a case in which the mother, who was a very intelligent woman, was quite certain that there was a boot button up her child's nose, because the child had been playing with one which could not afterwards be found. Two days after that the discharge in the nose began, and her story turned out to be correct, for the boot button was found to be in the nose.

Then, I think the *duration* of the condition may be a certain amount of help to you. For instance, *rhinitis* associated with adenoids has generally gone on a long time, and a great many of these cases are chronic. But some cases are much more acute. *Congenital syphilis* is acute, although it may last a long time. It is acute in this way: that the child will be brought for advice comparatively early in the trouble, because of the serious effect of the snuffles; the nose being so blocked up that the child cannot suck, and it is first brought for treatment probably for that reason. When *diphtheria* affects the nose it has probably lasted only a few days before the child is brought for treatment. Again, if the child has been *infected at birth* the disease is generally an acute one presenting alarming symptoms, and in that case the infant is brought early. *Atrophic rhinitis* has probably lasted for months, or it may be for years, before the medical man sees it. With regard to *tubercle*, nothing can be said, as the cases vary so. The presence of a *foreign body* is often associated with discharge from the nose for a long time—months or even years. I removed a mass of macerated newspaper from a child's nose some time ago; it had been there seven or eight months, and the discharge had lasted all that time.

Next we come to the *character of the discharge*. That is of much more importance than the duration, because one can recognise certain definite varieties in the discharges which come from the nose. For instance, in some cases the discharge consists of thick muco-pus, which does not come away, but blocks up the nose so as to lead to nasal

obstruction. That occurs most frequently in connection with *congenital syphilis*, and the *rhinitis* associated with adenoids. In many cases the discharge is distinctly blood-stained. It is usually so in *diphtheria*, and it is nearly always so in the case of *foreign bodies*. In tuberculous disease of the nose sometimes the history is that there has been a discharge of blood and pus from the nose. Then, again, in some cases of congenital syphilis, where the disease of the nasal mucous membrane has led to actual ulceration a blood-stained discharge will be present. We now come to pus. The discharge of pure liquid pus, that is to say pus which is not much thickened from admixture with mucus, is not very common, but it is found in certain conditions. Sometimes there will be a distinct purulent discharge in connection with a *foreign body*. But of course the condition which is associated with a discharge of real canary-coloured pus is *sinus disease*. Sinus disease of any kind is extremely rare in children, but a number of cases have been described, and the pus in such cases is commonly yellow and liquid. Moreover, in connection with sinus disease the pus has generally a peculiar odour, which, as you probably know, is associated with antral disease and frontal sinus disease. In other cases the discharge will have other characters. In *atrophic rhinitis* the discharge will consist partly of a watery mucoid fluid, and partly of large brownish or greyish green plugs which periodically come away from the nose. Atrophic rhinitis moreover is associated with the symptom known as *ozæna*, the peculiar disgusting sweetish odour which is noticed in this disease. Sometimes the discharge from the nose is watery in character. There are undoubted cases in which cerebro-spinal fluid does escape from the nose, and several of these cases have been described by Dr. StClair Thomson and others. A watery discharge from the nasal passages also occurs in connection with polypoid disease of the nose. In children, however, polypi are so rare as to be almost unknown.

The next thing I thought we might consider is the question as to whether the discharge is *unilateral or bilateral*. That is a very important point, because it enables one at once to distinguish certain conditions. For instance, if you see that the discharge is entirely limited to one side of the nose, it is almost certain to be due to a *foreign body*, because this is by far the commonest cause.

In only two cases have I seen anything else as the cause, and those were two cases of *unilateral nasal diphtheria* which I shall refer to again later. I have also put down in my list *sinus disease*, because although extremely rare in children there are a few cases recorded. If then the discharge is unilateral you should always suspect the presence of a foreign body, in most cases your suspicion will turn out to be well founded.

Now as to the *physical examination* of the nose. I need hardly say that examination of the interior of the nose in children is very difficult. That is so first of all because the parts are so small, and secondly because the children are generally frightened at the speculum and dark room. It is certainly difficult in many cases to get a good view. I think you can often get a comparatively good view of the interior of the nose in children by a very simple method, namely, by making use of a probe and not of a speculum at all, and using direct illumination instead of the forehead mirror and reflected light. If you place the end of a probe within the margin of the ala, and lift it well up and out, and at the same time elevate the tip of the nose, you can quite easily explore the inferior meatus and its boundaries, although you may not be able to see the middle meatus and the parts above. Actual examination through the anterior nares in that way will give a great deal of information. Probably you would not be able to diagnose the first three conditions in the table by this means only; but in the case of *diphtheria* you may see the membrane present, although there are some cases of nasal diphtheria in which no membrane is seen at all.

In the fifth cause in the list you would see some purulent discharge or muco-pus in the nose, with possibly some ulceration, and also a good deal of swelling and redness of the skin of the nose itself. The appearance in *atrophic rhinitis* is quite typical, and one can practically always diagnose it by inspection of the nasal cavity. In a case of this disease, if it has continued for any length of time, the first thing that will strike you is the large size of the nasal cavity, owing to the fact that more or less atrophy of both the bones and the mucous membrane has taken place, so that there is a great deal more room in the nose than there should be. The next point is that the walls of the nasal cavity are always covered over with greyish or greenish

crusts. You see these crusts situated on all the prominences of the nose, most frequently on the septum and on the inferior turbinated body. The appearance produced by the large nasal cavity with crusts over the surface is quite special to this disease.

With regard to *tubercle*, you may see definite ulceration, that is the most common thing in children in connection with tuberculous disease of the nose; or you may see perforation of the cartilaginous portion of the septum. In the only case of the disease that I have seen in a child, there was a perforation of the septum. Occasionally you may see a definite tumour, for quite large tumours may be caused by tubercle, and some tumours of fair size have been described in children.

Next we come to *foreign bodies* in the nose. In such a case inspection of the nose will usually reveal the presence of the foreign body, because it is nearly always situated in the inferior meatus; in a few cases it may not be possible to see it, because of the swelling of the parts around.

With regard to sinus disease, physical examination is most important, as it may, even in a child, enable you to make a correct diagnosis at once. The particular point to note is the position of the pus, which in disease of the antrum of Highmore or of the frontal sinus is always situated in one place, namely, above the inferior turbinated body; and you will therefore see it flowing over the upper surface of the inferior turbinate towards the septum. This is, of course, due to the fact that both the antrum and frontal sinus communicate with the middle meatus of the nose.

Other Symptoms Present.—These are very important, for in nearly all these cases there are associated symptoms in addition to the nasal discharge, and it is to these associated symptoms that one has to turn for the clue to the diagnosis. For instance, in No. 2 of the table there would be associated symptoms peculiar to adenoid growths of the naso-pharynx, *e. g.* nasal obstruction, mouth breathing, and perhaps deafness, either constant and associated with middle-ear disease or occurring in connection with the catching of colds.

In *congenital syphilis* no doubt there will be an associated rash in some part of the body, or other lesions may be present.

With regard to *diphtheria*, in the severe cases,

the most marked feature will be the pallor, just as it is so marked in most cases of faucial and laryngeal diphtheria; and you will also find that there are enlarged lymphatic glands in the submaxillary and deep cervical regions. In the mild cases, however, there will be no symptoms beyond the nasal discharge.

With regard to *infection at birth* there would not be much to be learnt from associated conditions, nor in *atrophic rhinitis*.

Very probably nothing further will be noticed in *tubercular disease* of the nose. There may be, however, other tuberculous lesions in the body, such as enlarged glands in the neck—and that is the most common thing associated with tubercle of the nose,—or there may be bone disease, or even lung disease. There will be no symptoms particularly associated with *foreign body* in the nose, and nothing special more than I have mentioned in connection with *unilateral diphtheria*.

Bacteriological Examination.—This is of great importance, and I want to lay great stress upon it, because it is not always carried out when it should be. *In any case of doubt as to the cause of discharge from the nose you should take a cultivation from the discharge.* By this means I have been able to detect a number of cases of nasal diphtheria, and the importance of that from the point of view of spreading infection cannot very well be exaggerated.

Those are the chief points in connection with diagnosis. I think I will occupy the rest of the time by going into a few of the special features of each of the conditions that I have put in the list of causes, and will start with the bilateral group.

No 1 I need not say anything about. But with regard to the rhinitis commonly associated with adenoids it is a very frequent trouble in children, quite apart from the other symptoms of adenoids, and therefore it is of extreme importance always to carefully examine the child for adenoids in all cases of discharge from the nose. Quite a number of cases come up every day to the out-patient department, in which there is a chronic discharge from the nose, and often excoriation around the nostrils. Although other symptoms of adenoids are usually present, such as nasal obstruction and mouth-breathing, this is not always the case, the rhinitis being sometimes the only symptom noticed. In many of these cases you will find

that there is a distinct adenoid cushion lying on the posterior wall of the naso-pharynx, opposite to the posterior nares. You may find such a condition present in some children who are not suffering from a nasal discharge in this way; nevertheless I am sure that when there is persistent rhinitis, this adenoid cushion is largely responsible for it, for if you remove the adenoids the nasal discharge nearly always clears up. There is one point I might mention here, namely, the best way to examine a child's post-nasal space. I have often seen students examine for adenoids with the patient standing facing them. That is not the most satisfactory way to do it. If you stand the child in front of you, but with its back towards you, it is perfectly easy to examine the naso-pharynx. In the first place the finger is curved in the right direction, and secondly, the child does not realise what is about to be done, and so is not frightened. You now ask the child to open its mouth; the right index finger is then immediately passed into the naso-pharynx, while the left index finger presses the cheek in between the teeth, so that you cannot be bitten. The examination can in this way be made in a very small fraction of a second, and quite satisfactorily. If, on the other hand, the child is facing you, it can see the finger coming, and hence there is probably a scene.

With regard to the other symptoms and the treatment of adenoids, I do not think I need go into that further.

We next come to the consideration of *congenital syphilis*. A very large proportion of cases of congenital syphilis are associated with so-called snuffles, namely rhinitis; but the exact lesion which occurs in snuffles I do not think is very well known. I have found only one reference to actual post-mortem examination, and that is in Ashbey and Wright's 'Diseases of Children.' They describe an actual case in which a child seven weeks old died with a definite rash and a history of snuffles for a week, and they say that "the whole of the mucous membrane of the nose was in a foul, almost sloughing condition, the surface being dark coloured and covered with muco-pus." There is, then, an acute inflammatory trouble, and later sloughing of the mucous membrane and bone infection, resulting in the formation of sequestra, which sometimes occurs. Apart, however, from the one case above referred to, I have been unable to find any account of the lesion which takes place

in the nose or nasal mucous membrane in snuffles.

Now we come to *diphtheria*. Nasal diphtheria is a very important disease in this way, that it has two distinct forms. In certain very bad cases of diphtheria which affect the throat and larynx, there is a certain amount of nasal diphtheria associated with it. These are generally very bad cases, and the majority of them are fatal. There is no difficulty about the diagnosis in a case like that. But there are other cases of nasal diphtheria of a different kind, and they are remarkable on account of the mildness of the symptoms. In some cases, indeed, there are no symptoms at all beyond a discharge from the nose. The child may seem well, and yet you will find on further examination that there is a large amount of membrane in the nose; or, again, there may not even be membrane present, but you will get a pure cultivation of the Klebs-Loeffler bacillus on taking a swab from the interior of the nose. I think these cases are extremely important, because, owing to the fact that they are not really ill, the patients go about spreading the infection. I have here notes of two cases of nasal diphtheria limited to one side of the nose, in which I am sure we should not have made the diagnosis had it not been for the regular bacteriological examination of all doubtful cases. I will read you the notes of these cases.

The first was a boy five years old, who was taken to the throat department of Guy's Hospital on July 28th, 1899. For ten days he had had a blood-stained discharge from the right nostril. The boy was otherwise in perfectly good health, and had not at any time seemed to be ill in himself. No history could be obtained of sore throats in the family or in the same house. On examination the right nostril was seen to be blocked by a dry crust. On removing this slight bleeding occurred, and a whitish grey succulent substance, very like a mucous polypus, could be seen. Portions of this were removed and proved to be oedematous membrane. The other side of the nose and the fauces were quite healthy. A blood-serum tube was inoculated from some of the membrane, and after twenty-four hours incubation a pure culture of Klebs-Loeffler bacilli was obtained. That is a very important case from the point of view of the spread of infection. This child was apparently well, but there was definite diphtheria on one side of the

nose. There was no history of diphtheria in the house, and therefore until a bacteriological examination was made there was great doubt about the diagnosis.

The second case was almost identical with the first. The child was seven years old, and was taken to the throat department of Guy's Hospital on August 11th, 1899, suffering from repeated hæmorrhage from the left nostril, of a week's duration. He was otherwise in perfect health, and as in Case 1 no history pointing to diphtheria could be obtained. On wiping away the dried blood from the left nostril slight bleeding occurred at once, but some white membrane could be seen in the nostril. This was of an opaque white appearance, and not gelatinous, as in Case 1. The other side of the nose and the fauces were healthy. A cultivation was made from the membrane, the result of which showed the presence of Klebs-Loeffler bacilli.

These were the first two cases of unilateral nasal diphtheria that I came across, and I published them to draw attention to their great importance from the point of view of infection. They appeared in the 'Lancet' of September 16th, 1899.

We now come to the question of *infection at birth*, presumably from some infective condition of the vagina. I do not think this condition has been very often described, although it is one which is well recognised as far as I am concerned. It is much more common to get blennorrhœa as a result of infection in this way, but there is no doubt that infection of the nasal mucous membrane does occur as well, or in some instances separately. The first case in which I learnt this fact was a very interesting one from the point of view of history. Three years ago two babies, twins, were brought here one morning, one suffering from blennorrhœa and the other from acute rhinitis. The nose in the one case was very much like the eye in the other case. The soft parts were swollen up and red and oedematous, and there was a considerable amount of purulent discharge. In the other baby one eye was greatly swollen, there was great chemosis, and it was obviously a case of blennorrhœa neonatorum. So I naturally concluded that the nasal case was one of infection, and since that time I have come across other similar cases. The affection is a somewhat acute one, as shown by the fact that in most of the cases the discharge has

been blood-stained, and therefore probably ulceration was present. I think it is wise to treat it actively, so as to try to stop it as soon as possible.

Atrophic rhinitis in children is a very rare disease, although it is comparatively common in young adults. However, there is a fair amount of evidence to show that certain cases start in childhood and persist throughout life, and this is particularly the case where there is a strong family history of atrophic rhinitis. The only child I have seen with it was a little girl of five, whose mother was suffering from the same complaint. She had well-marked atrophic rhinitis of a year's duration. The nasal cavity was large; there were numbers of dark greenish crusts all over the mucous membrane; and there was also the usual smell or *ozæna* which I have mentioned. The essential point in the treatment is to keep the mucous membrane as free as possible from crusts by the use of sprays or douches of alkaline lotion. The object of an alkaline lotion is to dissolve the mucus and remove the crusts. It is useful to add a small percentage of carbolic acid to the lotion, as that acts as a mild antiseptic and stimulant, and also helps to dispel the disagreeable odour.

With regard to *tuberculous disease* of the nose in children, that is also a very rare condition; quite a number of cases have however been described. I have a collection here, in a paper which I wrote on tuberculous disease of the nose ('Guy's Hospital Reports') of most of the cases which have been published. There are exactly one hundred. Out of that number there are seven in which the disease occurred in children under the age of twelve. In one case, in a small boy, at the age of a year, there was perforation of the septum, with ulceration and small nodules all round. Another case was a child aged eight months, in which nodules are described as being situated on the outer wall of the nose chiefly. That child died soon afterwards of tuberculous meningitis. There is another one, a boy aged twelve, in whom the nasal disease was associated with enlarged and caseous cervical glands. Here is a note of a case aged nine, in which there was a tumour situated on the right side of the septum, and which later on led to perforation of the septum. In that case healing took place. There are in this table other similar cases which I need not detail to you.

Tuberculous disease as it affects the nose is

rather different to tuberculous disease anywhere else, in that it often gives rise to large tumours. I remember a woman who came up to the throat department at Guy's Hospital with a tumour, which had destroyed the septum and was distending both nostrils. It was the size of a walnut. It was very vascular, and associated with frequent hæmorrhages, and I think nearly everyone who saw it thought it was sarcomatous in nature. However, before doing anything radical we cocaineised the surface, removed a small piece, and subjected it to microscopical examination. It turned out then to be tuberculous granulation tissue; and there are a number of cases described which illustrate the same fact, namely, that tuberculous disease in the nose may lead to the formation of quite large tumours. In the second place we have to remember that there may be ulceration and destruction of the cartilaginous septum. Supposing therefore that a child is brought to you suffering from a distinct vascular tumour in the nose, probably of the septum, although it may at first sight appear to be a case of malignant disease, there is a possibility that it is tuberculous in nature. Before carrying out any radical treatment in such a case, have it examined microscopically, in which way you can make sure what the nature of the tumour is. If it turns out to be tubercle, efficient treatment consists in thoroughly curetting the whole of it away, the little bleeding that generally ensues being easily stopped by pressure. Finally I might mention that cases of maggots in the nose, or myiasis, have been known in England, although it is much more common to encounter them in the West Indies, and in South and Central America. It is due to the fact that a fly actually lays its eggs in the nasal cavity during sleep. After a day or two these hatch, and the maggots set up a very acute rhinitis indeed, which is associated with ulceration, and sometimes with perforation of the septum; or it may even result in the formation of abscesses outside the nose.

The diagnosis is generally readily made by examining some of the discharge which comes from the nose, when the maggots will be seen.

There is great difficulty in getting rid of them, the only satisfactory plan being to kill the maggots first, and then wash them out. So far the vapour of chloroform has been found the most useful means of carrying this into effect.

We now come to the causes of *unilateral* discharges. I think the only one I need go into at all fully is foreign body. I have already mentioned unilateral diphtheria in connection with nasal diphtheria generally. Foreign bodies are often put into the nose by children, and there are a considerable number of cases of this sort brought here. Next to the rhinitis associated with adenoids I think it is the most common cause of nasal discharge. The presence of a foreign body in the nose is nearly always associated with nasal discharge. Usually it has been going on for some time, frequently for some months, and it is commonly blood-stained, and is also associated with excoriation of the nostrils and redness and cracks in the mucous membrane. On examining the interior of the nose you will generally see the foreign body lying in the inferior meatus. Occasionally, however, you will not see the foreign body at all, as happened in that patient I have mentioned in which some macerated newspaper was in the nose. I did not succeed in seeing that for a long time, and it was only after repeated douches that I saw part of this paper in the inferior meatus and was able to remove it.

With regard to the treatment of foreign bodies in the nose, all sorts of plans have been described for removing them. Some people use air in various ways. If the foreign body be in the left nostril one plan is to insert the nozzle of a Politzer bag into the opposite nostril and blow, the idea being to blow the body out through the anterior naris. That may be achieved, but in doing so there is a serious danger, and it consists in this: that supposing the foreign body is impacted in the left nostril and completely blocks it, as it probably will do, owing to swelling of the mucous membrane round it, you will be pumping air under pressure into the naso-pharynx, and at the same time you may inflate the middle ear. As this foreign body has been for some time in the nose, probably the post-nasal cavity will be infected, and there will be considerable danger of setting up some middle-ear disease as a result of blowing infective matter along the Eustachian tube. Other methods of inflation are used, but I do not know how commonly.

Another plan is to use warm lotion, injecting it into the nostril, either on the same side or the opposite side. If it be injected on the opposite side the notion is to wash the body forwards and out of the nostril. If it is injected into the side on which the foreign

body is, the object is to wash it into the naso-pharynx. One objection to that is that you may wash the body into the pharynx, and it may then become impacted in the air-passages.

There is another very simple way of removing foreign bodies, which I always make use of, and I have never had any trouble in getting a foreign body out of the nose by this plan. It depends upon the fact that the anterior wall of the nose is a good deal in front of the floor, where the foreign body nearly always lies. You stand behind the child, and take a probe and pass it towards you along the floor of the nose, by which means it is possible to displace the foreign body upwards. Then, by using the anterior bony margin of the floor of the nose as the fulcrum, the foreign body may be pushed by the probe upwards and forwards. The foreign body is now situated between two inclined planes which diverge downwards and are formed by the probe and the anterior wall of the nose, and therefore it can only move in one direction, namely, downwards. Pressure exerted by the probe causes this downward movement, and so the body escapes. In this way I have removed all sorts of things. Some of the most common articles placed in the nose are black boot buttons, pieces of slate pencil fruit stones, and peas. The other day we had a good deal of trouble in getting a large French bean out of the nose, because it had been there some time. In another instance I removed a large piece of sponge. Last summer a child was brought up with a lot of macerated brown paper in its nose, and that came out easily. I strongly recommend you to make use of this method first, because it is very simple and very efficient. Stand behind the child and pass the director straight in through the nostril and press it against the upper lip, then the foreign body nearly always falls out on to the floor.

Cancer Distribution and Statistics in Buffalo.—J. P. Lyon states that the house distribution of cancer shows an area of marked concentration in the German wards. Cancer is more frequent among the foreign born, and particularly the Germans, than the native born. The cancer rate of foreigners in general in Buffalo is 4.59 times the rate for the native born.—*American Journal of Obstetrics*, September, 1901.

A DEMONSTRATION OF CASES AT CHARING CROSS HOSPITAL.

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CEREBRAL ABSCESS.

THE first case I want to show you is one of cerebral abscess. There are many points of importance to be observed in connection with this patient's condition. Before demonstrating her case to you I will give you briefly the details of the history. The patient is a girl *æt.* 26. She was admitted into the hospital with the following history:—She had had a discharge of pus from the right ear since infancy. The discharge was not constantly present, but it came on at intervals. At the age of twelve she had scarlet fever and diphtheria, and these maladies of course markedly increased her ear trouble. She had also diphtheritic paralysis of the palate and ciliary muscles. The discharge has always come from the right ear. The history of the present attack is as follows: about three weeks prior to admission the patient was suffering from earache. She had slight loss of vision, and she complained of some strange feeling in the eyes. The earache became extremely severe; so much so that she had to leave her situation. Complaint had been made of intense headache for something like nine days before she was admitted to the hospital. The headache continued and got worse until practically it became unbearable. She was admitted into the hospital late one night, in a partially unconscious state. A very noticeable point about her was that her pulse was only 44 beats per minute. Mr. Bonney, the house surgeon, from a consideration of the symptoms I shall detail presently, felt pretty certain that she had intra-cranial abscess, and he sent for me about midnight. When I came down I was at once struck with the fact that she had an extremely slow pulse, which, as I have said, was 44. On examining her I found she had slight exaggeration of both knee-jerks, and slight commencing ankle clonus. Tapping over the skull on the right side caused the patient to wince while it was being done, though she was too insensitive to complain of pain. On examining her eyes I could find no

strabismus, but the right pupil was rather more dilated than the left. She had commencing double optic neuritis. We could not get her up so as to see whether she staggered, because she was so drowsy, and she lay almost as if she were dead. Thus, the really salient symptoms we had were agonising headache for nine days, discharge from the right ear for practically all her life, slight commencing optic neuritis, and the history that she had vomited. The vomiting, however, was less than one usually meets with. There was nothing characteristic about her temperature, which stood at about 100°. The optic neuritis and slow pulse indicated increased intra-cranial pressure. With the discharge from the ear the increased pressure could be due to two causes only; one was meningitis, and the other cerebral abscess. It did not seem to be due to meningitis in this case, the only thing pointing to meningitis was the fact that she had some rigidity of her neck muscles. I had no hesitation in making the diagnosis that this girl was suffering from intra-cranial abscess of the right side, due to ear disease. The thing which gave me great difficulty was to ascertain whether there was in this case an abscess of the temporo-sphenoidal lobe, or an abscess of the cerebellum. It was Toynbee's rule that if you have disease in the tympanic cavity and an intra-cranial abscess, the latter will be an abscess of the temporo-sphenoidal lobe, and if the disease be in the mastoid cells the abscess will probably be in the cerebrum. This rule is not reliable, because it is found that in disease which has lasted almost a lifetime, there will probably be suppuration both in the tympanum and in the mastoid cells. It is a somewhat interesting point that this is the third successive case of cerebral abscess upon which I have operated successfully, and yet in every case I have first attacked the wrong part of the brain. In this girl's case there was one symptom that is supposed to be of importance, and which I have found very fallacious. It is this: It is said that in cerebellar abscess the grasp of the hand is weakened on the same side as the abscess. But in this case I and the house-surgeons and house-physicians were quite certain that the grasp of this girl's right hand was markedly weaker than the left. Therefore I concluded that the balance of probability lay in favour of cerebellar abscess. I, therefore, trephined the right cerebellar fossa. Then I inserted

a grooved needle and exploring syringe in two or three directions, but nothing came away. Therefore I took the disc of bone which I had trephined and put it back; you can feel that it now fills the gap. I next trephined one and a quarter inches above the external auditory meatus, and perhaps one quarter of an inch behind it. I inserted a needle, and immediately foetid greenish pus came out, rather more than two ounces in quantity; with it were some blood-red sloughs of brain tissue. I gently irrigated the abscess cavity and inserted a drainage-tube. An interesting point was the very slow recovery of the patient. The temperature was 100.2° , and the pulse 44 before we operated. The pulse continued slow for, at least, a couple of days—practically forty-eight hours—after the operation, and then it rose to 62. The patient remained at this time practically unconscious. That is really what one must be prepared for. There was an abscess involving the greater part of this temporo-sphenoidal lobe, and the brain substance around the abscess was not healthy, but in a state of septic cerebritis, and though you remove the pus, of course, the conditions do not return to the normal immediately. For some days she had a copious discharge of pus and some sloughs. She was delirious for certainly days after the operation, so that, notwithstanding the close watching which was kept up, she twice managed to pull off her dressing and remove the tube. Even in spite of this, no appreciable harm was done. The house-surgeon reinserted the tube and redressed the wound carefully. The girl is now practically well. She had still some slight discharge from the ear some little time after the operation, and on closely examining her ear I found that the meatus was blocked with aural polypi, of which I removed four. Now she has a perforation in the membrana tympani which discharges a small amount of pus, but the quantity is nothing like what it used to be. She had, for about a week after the operation, very little ordinary cerebral control—if one spoke roughly to her and said she must not pull her tube out again, she simply wept silently; she was violent at times, and did not give rational answers to questions.

There are certain general points about abscess of the brain which I think are worth mentioning. In the first place, we know that an abscess of the brain is practically never a primary disease; more

than half the cases are directly due to ear disease, and about half of the remaining ones are due to traumatism, *i.e.*, to a compound fracture or concussion. Probably the greater number of the remaining quarter are pyæmic. Of course pyæmic abscesses of the brain are, as a rule, multiple. Abscess of the brain due to aural disease and due to traumatism or to extension of pyogenic processes from the nose or naso-pharynx, is usually single. Of course operation in multiple abscess of the brain is always contra-indicated from the nature of the case operative interference being hopeless. On the other hand, operation in well-chosen cases of single brain abscess is most favourable. A point which has strongly impressed itself upon me is that one must not delay operation in cases of brain abscess. I need hardly point out to you that in this case there had been delay almost as long as could be with recovery; indeed, we thought the limit of safety had been transgressed, because of her pulse being only 44 when she got to the hospital, and because she was practically unconscious. Sudden death is very apt to occur in these cases. One case in this hospital impressed me very strongly about a year ago. A man was admitted with certain signs of cerebral abscess. I need not go fully into that case now, but we considered the case very carefully on Saturday afternoon, and it was decided that we should wait for more localising symptoms. There was no localising symptom, however, as there was no localising symptom in the present case. It was because of the absence of localising symptoms that in this present case I first attacked the cerebellum, and it was only after failing to find pus there that the temporo-sphenoidal lobe was explored. In the case of a year ago there was only agonising headache, but no optic neuritis, though there was a little blurring of the discs and a history of ear discharge. I thought that in all probability it was abscess of the brain. I took advice in the case from my colleagues, and it was considered that it would be hardly justifiable to explore the cranial cavity in the absence of more distinct localising symptoms. I came down to the hospital on Sunday afternoon and said to the house surgeon: "I have come down to explore that brain as I do not feel easy about it." He replied, "The man died suddenly about five minutes ago." Now, cases like that make one feel that once the diagnosis of intra-cranial abscess is

made, the sooner operative treatment is undertaken the better. As soon as there are any definite symptoms pointing to the increased intra-cranial pressure, and you suspect an abscess, it is wise to explore at once. We seldom get the pressure symptoms so marked as they were in the present case with a pulse of only 44. As I have said, one must not expect the opening of the abscess to be followed by immediate recovery. Frequently the patients lie for some days afterwards almost like dead people; the reason being that although the pus has been evacuated you still have the brain in the immediate neighbourhood cedematous and infiltrated, and it is really in a condition of septic cerebritis. It is an unfortunate fact that localising symptoms in cerebral abscess are usually absent. It is very seldom we get an abscess in the brain which involves the motor area or even the speech centre. A symptom of very considerable value is percussion of the head. If percussion of the skull elicits pain, this will sometimes strongly support the diagnosis of brain abscess. You will generally find that the point of maximum tenderness is directly over the abscess. Our patient tells us now that her brain feels all right, except that it is a bit weak. It is now eight weeks since the operation. She believes her condition gets better every day.

MULTIPLE FOCI OF TUBERCULOSIS.

Here is a child æt. 1½ years, the subject of tuberculosis. It is interesting because it presents, as so many of these tubercular children do, multiple foci of tuberculosis. He has a finger and a toe presenting very markedly the condition known as tubercular dactylitis. I ask you, also, particularly to notice this mass of subcutaneous tubercle. Tubercular dactylitis has very little special about it; it is tubercular disease starting in the medulla or marrow of one of the phalanges. As the tubercle increases it thins the bone and distends it, and you thus have produced the regular globular shape which is shown in these specimens. In addition, the child presents a subcutaneous mass of tubercle, which has just suppurated. When I saw the child about a week ago, this last felt like a hard button, almost exactly like a hard chancre underneath the skin. That is a condition which you frequently find in children, but very rarely in adults. It is what the old

surgeons used to call a scrofulous gumma. You know how extraordinarily tubercle picks out certain parts at certain age periods. In young children under ten years of age tubercle is very apt to affect the subcutaneous tissue in the form of so-called scrofulous gummata of subcutaneous tissue. The prognosis of tubercle in the subcutaneous tissue, however, is very good. The mass of tubercle softens, and either bursts or becomes absorbed, leaving a depressed scar. The treatment of these cases of tuberculous dactylitis I have shown you on previous occasions. The treatment I shall adopt in this case is to make an incision into the bone, scoop the whole medulla out, and then pack it with decalcified bone chips, leaving simply a thin scale of bone. You will observe the typical globular shape. In the early stages this condition is most favourable to treatment. I told another patient to come up to-day, but I am sorry to say he has not done so. In that case there were three phalanges affected, from which I scooped out the whole medulla and packed the bony cavities with decalcified bone chips. This child now uses all three fingers almost perfectly. It is an infinite improvement on the old treatment of amputating the finger for tuberculous dactylitis. In the great majority of cases there is no need to resort to such mutilation.

(To be concluded.)

An Atypical Case of Addison's Disease.—

At a recent clinic at the Beaujon Hospital, Prof. Debove demonstrated an atypical case of Addison's disease in a man of sixty-three ('L'Indépendance Médicale,' June 12th, 1901, No. 23). He has travelled a great deal, and has had dysentery and malaria. His face, hands, neck, and scrotum are dark brown, and the cheeks, lips, and conjunctivæ show brown spots. His spleen is normal. The skin has become hard, rugged, and dry; he is cold, and constantly exhibits "goose-flesh." This melanoderma began twenty years ago. In spite of the absence of all other symptoms, and the long duration of the pigmentation, Debove diagnosed Addison's disease, and ordered 10 to 40 cg. of suprarenal extract as treatment.—*Philadelphia Med. Journ.*, October 12th.

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* Specially reported for The Clinical Journal. Revised by the Author.

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A CLINICAL LECTURE

ON

INFANTILE SCURVY.

Delivered at the Hospital for Sick Children, Great Ormond Street, July 4th, 1901.

By ARCHIBALD E. GARROD, M.A., M.D.,
F.R.C.P.

LADIES AND GENTLEMEN,—I am fortunate in being able to bring to your notice this afternoon a most characteristic case of infantile scurvy, which was admitted to the hospital yesterday afternoon, and in which treatment has not yet had time to bring about any appreciable change.

The first point to which I would call your attention is the tenderness which is manifested by the crying of the child whenever it is moved, but which is not so marked in this instance as it often is in such cases. Not only does the child cry whenever it is moved or handled, but it will cry at the mere apprehension of being handled. It is, indeed, one of the most characteristic symptoms of this disease that the patient will scream when anyone approaches its cot, because it knows, by experience, the extreme pain attending any movement to which it may be subjected.

Associated with the tenderness is the immobility of the patient. You will notice particularly that the left leg of the infant is held absolutely still, and the immobility is often so complete, that it is quite a common thing for the mothers of such children to bring them up to the hospital with the story that they have lost the use of their limbs. There is, indeed, a sort of pseudo-paralysis, which must, of course, be most carefully distinguished from true paralysis of any of the varieties met with in young children. In the slighter cases the legs may be held drawn up, but in the most severe cases they lie everted as if all power were lost in them.

The next point to be noticed in this particular case, as in most cases of infantile scurvy, is the condition of the gums. The gum affection is not

absolutely constant, as I shall explain to you presently, but in the great majority of cases which are at all severe, it is present to a greater or less extent. In the slighter cases it may not amount to more than a purple discolouration around the teeth, and such discolouration may be present over teeth which have not actually been cut, but are on the point of eruption. When only a few teeth have been cut the affection is limited to the parts of the gums adjacent to them. If the teeth are not approaching the surface the gums will not be affected at all. It is very important to remember this fact because it may add considerably to the difficulty of diagnosis. In very young children, that is to say children of six or eight months, who have well-marked scurvy you may find that the gums are quite normal. In more severe cases the gums become raw and swollen around the teeth which have already been cut. In more severe cases still the gums become spongy, bleed on the slightest touch, or even without any provocation. In the particular case before you the gum phenomena are very well marked indeed. In the neighbourhood of the upper incisor teeth you will notice that the gums are spongy and ulcerated, and bleed on the slightest provocation. In fact, the bleeding from the gums was the first thing which called attention to the condition of the child, and you will not often see the phenomena better marked than in this case. In the most extreme cases, such as you will see described by Dr. Cheadle in some of his writings, the gums form bleeding, swollen masses which project between the lips. These extreme cases are very rarely seen at the present day, partly because we know more about the condition, and it is seldom allowed to go on to this stage.

A third conspicuous feature is the occurrence of swellings in the limbs around the long bones, and such swellings are present in this instance. They are most frequently met with around the bones of the lower limbs, and most commonly of all about the lower parts of the tibiae. The femora are very often affected, and in this case over the left femur there is a conspicuous and very tender swelling, and a second swelling over the sternal end of the right clavicle. You will observe that the skin which covers these swellings is not reddened, and when the hand is applied to them no particular sense of heat is experienced. Such

swellings over the diaphyses of the long bones are due to subperiosteal hæmorrhages, and I will presently show you some specimens which illustrate their nature.

There are various other symptoms of infantile scurvy which call for notice. Anæmia is common, especially in severe cases, and particularly, I think, in cases such as this, in which the periosteal swellings are most marked; in a word, in those cases in which large hæmorrhage has taken place beneath the periosteum. But such hæmorrhages do not account for the whole of the anæmia. In this instance the blood count is decidedly low, the red corpuscles being about 3,000,000, the white corpuscles about 11,000 in the cubic millimetre, and the hæmoglobin 34 per cent. There are no abnormal leucocytes. Dr. Thursfield, who made the blood count, tells me that this is a lower figure for the red corpuscles than he has usually met with in cases of this disease. In the descriptions of infantile scurvy, and particularly in the earlier descriptions, which were for the most part based upon very severe cases, you will find anæmia and a clay-like cachectic appearance described as characteristic features. In the milder cases those phenomena are not always well marked.

Owing to the fact that children with infantile scurvy are usually somewhat rickety, the disease is very often spoken of by the name of "scurvy rickets." In this case there is a certain amount of beading of the ribs, but it is not very extreme. Moreover the fontanelle is not nearly closed, and although the child is fifteen months old its teething is not very far advanced.

In cases in which a recent subperiosteal hæmorrhage has occurred, as in this instance, the temperature is often raised to 101° or 102° for a few days, but elevation of temperature is not by any means a constant phenomenon of infantile scurvy.

In the course of practice you will occasionally meet with infants who exhibit a marked degree of general tenderness, usually in association with rickets, but in whom the gum affection and other signs of infantile scurvy are wholly wanting. Dr. Cheadle has suggested—I do not know whether he was the first to suggest it—that there are cases of infantile scurvy in which such tenderness is practically the only thing observed, and which recover in the characteristic manner under antiscorbutic

treatment. The effect of alteration of diet in such cases, which is undoubted, appears to me to afford very strong evidence of the correctness of Dr. Cheadle's view, and as tenderness is usually the first symptom developed, it is in no way remarkable that cases should be occasionally met with in which the tenderness is the only symptom present, and in which suitable and prompt treatment may prevent any further developments.

In a second group may be included those cases in which a young infant, as yet toothless, has no gum affection, but presents tenderness and subperiosteal swellings. Such cases are sometimes rather puzzling, because one hesitates, in the absence of gum phenomena, to feel sure that these periosteal swellings are due to infantile scurvy. The effect of an altered dietary will soon show whether or no the correct diagnosis has been arrived at.

In a large class of cases—perhaps the largest of all—there is the general tenderness on handling, together with some degree of the gum affection, amounting to either a little purple discolouration over the on-coming teeth or purple discolouration and swelling around such teeth as have been cut. This is the form of the disease most frequently met with in ordinary hospital out-patient practice.

The periosteal swellings if present may occupy various situations. I have already mentioned that they are more common in the lower than in the upper extremities. I can recall one case in which there was merely general tenderness and a periosteal swelling over one humerus apart from any gum affection, the infant being toothless. The course of the case under treatment showed clearly that it was one of infantile scurvy. Swellings have also been observed over the scapula. In some of Sir Thomas Barlow's cases such swellings were present, and they have even been observed on the skull, on the clavicle as in this case, and on the ribs. Not unfrequently a peculiar and characteristic flattening of the front of the chest is present, due to a sinking-in of the sternum, as the result of fracture at the junctions of the ribs and costal cartilages. When present this is a very characteristic phenomenon of the disease, and it is one to which Sir Thomas Barlow was the first to call attention. The peculiar flattening of the chest referred to is not present in the case before you.

Hæmorrhages may also occur around the ribs near their junctions with the cartilages.

Again, hæmorrhages occasionally occur beneath the periosteum of the orbit, with the result that protrusion of the eyeball is suddenly developed. This is not simply a clinical curiosity, for it has been observed in a considerable number of cases, and has not unfrequently given rise to errors of diagnosis. Those who have written on scurvy rickets have described such occurrences, and a special paper on this subject, from the point of view of the ophthalmic surgeon, has been written by Mr. Holmes Spicer. In such cases, in association with the orbital hæmorrhage there may be hæmorrhage into the eyelid itself, leading to very considerable bruise-like swelling.

Cutaneous hæmorrhages are also met with. They are not common, but some petechiae or large bruise-like lesions are present in a certain proportion of these cases.

Hæmorrhages from the mucous membranes are not uncommon, the most frequent being hæmorrhage from the gums, which has been a marked feature in the present case, and which led to the child's being first taken to a dental hospital for treatment. Epistaxis may occur, and hæmorrhage from the bowel, and lastly hæmaturia. Hæmaturia is present in not a few of the cases. In one which was in Alice Ward a few months ago, it was a prominent symptom. The other phenomena of infantile scurvy were, in that case, comparatively slight; but the hæmaturia went on for some time, and curiously enough, when the child had an attack of an intercurrent febrile disorder—I think it was bronchitis—there was a return of the hæmaturia, though the scorbutic condition had been almost recovered from.

In connection with hæmorrhage around the long bones certain additional troubles may occur, the chief of which are fractures in the neighbourhood of the epiphyses. Here is a specimen from a case in which there was an immense hæmorrhage round the femur, and the epiphyses have become completely detached. The diaphysis separated from both the epiphyses lies buried in a mass of blood clot; and here is a second specimen in which, as you will notice, the lower epiphysis of the femur has become laterally displaced. An actual fracture may occur in the shaft itself, but that is not common.

The causation of the disease may nearly always be traced to some well-marked error of diet. In the majority of cases some artificial food has been given, and, in addition, either condensed milk, or milk which has been subjected to a considerable amount of preparation. The sterilisation of milk is certainly responsible for the development of a certain number of cases of infantile scurvy, and I think that the very widespread use of sterilised milk at the present day is responsible for some increase in the number of the slighter cases. I have also seen infantile scurvy with subperiosteal hæmorrhage in children who have been fed upon humanised milk, in the preparation of which sterilisation is one of the stages. In a considerable number of instances there is a history of feeding with condensed milk, but the most severe cases are met with amongst children who, for a time at least, are fed upon a diet containing no milk at all, on a starchy food and water, for example. Now and then you will meet with a case in which the causation of the trouble is by no means clear, and, indeed, the case before you affords an example of this, for the history is not such as one expects in a severe case of infantile scurvy. This child was never breast fed. At first it was given cow's milk and water, one third milk to two thirds water, and after three months it had barley water and boiled milk in equal proportions. After six months a malted food was added to the diet. He had been in the habit of having one and a half pint of milk *per diem*, and the milk was not allowed to boil for more than a minute or two. During fourteen days whilst the child had whooping cough malted milk was given, but with this exception his food has not varied. Hundreds of infants are fed in this way without developing any scorbutic symptoms, and it is difficult to make out what can be the special determining cause of the scurvy in this case.*

The next point which we have to consider is, in what relation the scurvy stands to the rickets. There can be little question that, as Sir Thomas Barlow maintained from the first, there is no essential connection between the two. A child may be

scorbutic but not rickety. Cases have been recorded of children who showed no signs of rickets, but in whom very definite signs of scurvy were present. The signs of rickets are often very slight. Thus we see that there is no intimate connection and no direct proportion between the two. The scurvy may be severe and the rickets slight, or there may be severe rickets with slight scurvy. And if you consider that both rickets and scurvy are in all probability diseases which result from errors of diet, you can quite readily understand that an error of diet which is likely to produce scurvy is likely on the way to produce rickets also, and this I take to be the cause of the so frequent association of the two conditions.

I have a set of specimens here, some of which are of considerable historical interest, which illustrate the morbid anatomy of infantile scurvy. The growth of our knowledge of the disease is intimately associated with this hospital. The first case in which the subperiosteal hæmorrhage was definitely demonstrated and described was under the care of one of our consulting surgeons, Sir Thomas Smith, and it was provisionally described under the name of "hæmorrhagic periostitis." Dr. Gee described some cases under the name of "periosteal cachexia." Afterwards Dr. Cheadle described some cases as illustrating the association of rickets with scurvy (I am only referring to those who have been connected with this hospital). Lastly, Sir Thomas Barlow published his classical paper, in which he brought together the clinical features of the disease and the anatomical phenomena, and gave the whole question so definite a standpoint, that on the continent the name of "Barlow's disease" is almost universally employed to designate this particular morbid entity. I should add that the earliest description of cases of this kind was given in 1857 by Möller, of Königsberg, who described them under the name of acute rachitis. Various other examples of acute rickets were afterwards described on the continent, but it was really the investigation I have mentioned which gave us a definite knowledge of this as a distinct disease. These are Sir Thomas Barlow's original specimens, which illustrated the paper to which I have referred. There are other specimens here which illustrate certain particular points, such as hæmorrhage around the ribs and hæmorrhages about the scapula. This is a specially interesting specimen

* Under the ordinary antiscorbutic treatment the child made a rapid and good recovery. The tenderness and gum affection soon disappeared, but the absorption of the swelling around the femur was naturally a matter of longer time.

It is from a case of very long standing, in which, without the absorption of the effused blood-clot, a certain amount of repair has gone on, and you will notice that the stripped periosteum has itself become the seat of ossification, so that we have first a layer of bone representing the periosteum, then organised blood-clot, and lastly the diaphysis of the femur itself.

To turn now to the pathology of the disease. The name which I have used throughout this lecture rather begs the question. In this country the name of infantile scurvy, or very frequently of scurvy rickets, is generally used to designate the disease of which I am speaking. The arguments in favour of regarding this as a form of scurvy are fairly strong, and they have not met with any serious question, either here or, as far as I know, in the United States. But on the continent, on the other hand, the scorbutic origin of these cases is by no means universally accepted. I have referred to the common use of the name Barlow's disease for infantile scurvy. In the use of that name there is something implied beyond the mere compliment to Sir Thomas Barlow. It is, indeed, employed as a non-committal name, because those who use it are, for the most part, by no means convinced that it is the same disease as we know by the name of scurvy in adults, which formerly occurred very largely on board ship, in prisons, and occasionally in other places where bad diet conditions prevailed. The earlier observers—Möller and those who called the infantile malady acute rickets—regarded it as a peculiarly acute form of rickets. Then was developed the scorbutic view, which was first seriously started by Dr. Cheadle, and which was adopted and worked out to a great extent by Sir Thomas Barlow. That view is chiefly based upon the facts that in this disease, as in scurvy, the gums are very conspicuously affected; and hæmorrhages are very common phenomena. Moreover, like adult scurvy, it is a disease depending on something wrong in the feeding of the patient, and, above everything else, it is a disease which is rapidly cured by the remedying of such errors of diet. The arguments used by those who doubt the scorbutic origin of the disease are somewhat of this kind. That those who have had opportunities of observing epidemics, of land scurvy especially, have observed an immunity on the part of young infants, an immunity which may perhaps be explained by the

fact that the infants may not share in the particular dietary privations which lead to the outbreak of the scurvy in the adults. In the second place, it is said that sponginess of the gums may be present in other hæmorrhagic diseases besides scurvy. That is perfectly true. A stronger argument is based on the fact that although hæmorrhage under the periosteum has been observed in adult scurvy, it is not by any means a common event in that condition. On the other hand, cutaneous and subcutaneous hæmorrhages, which are very common in adult scurvy, are comparatively uncommon in this infantile disease. To this it may be replied that in young children the parts in most active growth are to some extent in a hyperæmic condition, and are therefore the most likely to be the seats of hæmorrhage when any hæmorrhagic tendency is present. This is, however, a theoretical answer, and it is not accepted as conclusive by the opponents of the scorbutic theory. Those who doubt the scorbutic nature of the infantile disease do not offer any satisfactory alternative theory of its pathology. An infective origin has been suggested, but this view has very few adherents. A very considerable number of authors hold that it is a form of rickets complicated by a hæmorrhagic diathesis. This it undoubtedly is, and the only difference between us is that we in this country are in the habit of regarding this hæmorrhagic diathesis in question as what is known as scurvy. We see, then, that the attitude of continental observers is rather one of scepticism than of offering any definite alternative explanation of the disease, and that is why such a name as Barlow's disease commends itself to them, seeing that it commits those who use it to no definite view as to the pathology of the malady. It seems to me that of the points in favour of the scorbutic nature of the disease, the argument based upon the effects of treatment is worth all the others put together. There can, indeed, be no question about the remarkable effect of suitable treatment in this particular disease.

To anyone who is acquainted with its symptoms the diagnosis of infantile scurvy seldom offers much difficulty. There are not very many conditions for which it can be mistaken. If there be the gum affection, general tenderness, and periosteal swellings, you cannot very well confuse the malady with any other. Of course, if there are

no teeth, and if there is no gum affection, but merely the periosteal swelling and the tenderness, the diagnosis is not so easy. However, there are not very many conditions in young children in which such tenderness is observed. I have already expressed my belief that such general tenderness, even when it stands alone, is usually due to the presence of infantile scurvy in its earliest stage. There is, however, one condition which resembles it in this respect, and that is epiphysitis due to congenital syphilis; that produces a similar tenderness, and the same kind of consequent immobility. It is a less common affection, but in that disease, as in scurvy, the mother frequently comes to one with the story that the child has lost the use of its limbs.

The prognosis of infantile scurvy is, on the whole, very favourable. The specimens before you show that it is not a disease that uniformly ends in recovery, and in the earlier days, before the nature of the condition was at all well recognised, and when its appropriate treatment was unknown, undoubtedly many of these children did die, and a certain number of them die even at the present day. There was a fatal case in this hospital within the last year, and it was the case of the child from whom this femur was taken. When a child has once reached the stage of extreme anæmia and cachexia, with very extensive subperiosteal hæmorrhages, whatever treatment you may adopt the case may have a fatal ending. Continental observers lay very much less stress on the curative effect of appropriate diet than we do here. That is very remarkable, because if there is one thing which appears to be established in the whole range of therapeutics, it is that in the slighter cases of this disease the curative effect of an appropriate diet is remarkable and rapid. Apparent failures of so-called antiscorbutic treatment may be explained in several ways. Undoubtedly we meet with failure in cases in which the disease has advanced too far before the treatment is commenced, and in which the child may succumb in spite of treatment. You may also meet with failures in the cases which are incorrectly diagnosed, and I think some of the cases recorded in which antiscorbutic treatment was not successful may be put into this class. But given a case which is not too severe, in which there are no very great subperiosteal hæmorrhages, and in which the child is not too exhausted

and anæmic, you may promise that in the course of a comparatively short time there will be a marked improvement, and even recovery. There is no disease which it is more worth your while to diagnose accurately than this, seeing that there is no disease in which appropriate treatment meets with a fuller measure of success. Yet it is a disease the characters and treatment of which are by no means so universally recognised as they should be.

Even if it be granted that the disease under consideration is identical in nature with the scurvy of adults, we do not arrive much nearer to a knowledge of its true nature, seeing that the pathology of scurvy is still very obscure. We do not know whether it is due to deprivation of some necessary element of diet, or whether it is due to the presence of noxious substances. Some of you may have seen a recent paper by Professor Vaughan Harley and Mr. Jackson, in which they conclude, from a number of experiments upon animals, that scurvy is due to eating meat which is not fresh; is due to the formation of poisonous substances in the preserved meat. That is to say that it was due to the presence of a positive noxious element, and not to the mere absence of some antiscorbutic constituent from the diet. The chief difficulty of accepting such a view lies in the fact that recovery so quickly follows the addition of certain substances to the diet.

In the matter of treatment, we are here in the habit of following the lines laid down by Dr. Cheadle and Sir Thomas Barlow years ago, and whether all that they prescribed is necessary or not, certainly that treatment produces the desired results. In the first place, if you can rely on your source of milk supply, unboiled milk is certainly preferable to boiled milk for such children. In the second place, raw meat juice is usually given, and it is believed to contribute very materially to the success which attends antiscorbutic treatment. The only drawback to the raw meat juice is the possibility of giving tapeworm to the child by its means. For that reason it is very advisable to strain it through fine muslin, which will arrest the tapeworm ova. Next, the juice of fresh fruits, such as oranges, may be given, it is palatable and seems to be useful in such cases. An important point may be referred to in connection with feeding on sterilised milk. You may think it necessary—and undoubtedly it is very often highly desirable—

that a healthy child should have its milk sterilised. Under these circumstances the risk of the development of scurvy may apparently be avoided by the addition of small quantities of fresh orange juice to the diet as a precautionary measure. Fresh vegetables have also been recommended in the treatment of scurvy, such as well steamed potato, run through a fine sieve and made into a sort of cream with milk.

Medicine occupies a quite secondary place in the treatment of this particular condition; in fact, in most cases one does not give medicine at all, unless there are symptoms present which seems to call for it. Under the treatment I have indicated, improvement begins in a remarkably short time. In forty-eight hours there ought to be definitely less tenderness than there was at the onset, and in the course of a week or ten days, unless the disease is very severe, the child will be on the highway to recovery. The rapidity with which the swellings of the limbs disappear is sometimes remarkable, but the actual absorption of blood clot beneath the periosteum necessarily takes some time.

There are other minor points in connection with treatment which call for mention. In severe cases, such as this, one must be careful not to run the risk of fractures near the epiphyses, and therefore the less the limb is handled, and the more gently it is handled when handling is necessary, the better. The child should not be moved about, except as far as is necessary for the sake of cleanliness. In the out-patient department, when one saw a slight case which it was not necessary to admit, one instructed the mother to carry it upon a pillow whenever it was necessary to move it about.

Lastly, there are the ordinary hygienic measures which should be employed. Fresh air and good hygienic conditions will help recovery; and when the child is getting better, cod-liver oil may be given; but medicines are not of any real value in the treatment of the scorbutic condition as such.

In conclusion, although infantile scurvy is by no means a common disease, you are at any time liable to meet with such cases in your practice. I shall be very glad, indeed, if anything that has been said this afternoon should help any of those present to recognise the condition, especially in its slighter forms, because the success which follows upon suitable treatment renders the early and accurate diagnosis of the malady a matter of great importance.

IMPAIRED VIRILE POWER.

By CAMPBELL WILLIAMS, F.R.C.S.

IMPAIRED virile power signifies a condition of decreased or deficient sexual potentiality occurring during that period of life when an average normal adult should be physiologically active. It implies a modification of a former existing standard and expresses a distinction to the state of impotency, such as results naturally at an approximate age, from advancing senility, or which is due to congenital defect or absence of instinct consequent upon arrested development of the higher cerebral centres or of the reproductive organs. Although the phenomenon may be the expression of some grave and progressive nervous degeneration, of some intercurrent malady, or of special organic deterioration with which we cannot cope, and bearing in mind that some cases of uncomplicated impairment do culminate in spite of treatment in premature and permanent impotency, nevertheless, a certain proportion of those affected are practicable of comparative or actual sexual rehabilitation. Particularly so when a functional or remediable element dominates the case. The psychological state induced or coexisting in the patient often forms a preeminent symptom. This is apt to be an ever present morbid consideration of the erotic disability, so that auto suggestion, either the result of conscious or unconscious cerebration, by generating strong descending inhibitory influences, may perpetuate the condition. These, as it were, hold an hypnotized manhood in abeyance, as with the paralysis of hysteria. It requires but a stimulus to once more set in motion the dormant but undestroyed mechanism. A goodly number of such cases are traceable to a continuation of an evil habit acquired during adolescence, or to excessive sexual indulgence. The standard constituting excess is a matter of individuality, for what is such in one person is not in another. A history or clinical evidence of an antecedent attack of gonorrhoeal prostatitis is not an unfamiliar occurrence. But with a certain percentage of those who suffer in this manner the event is not due to any fault of their own creation or control. Its production may be influenced by the presence of an inherited varicocele, causing testicular malnutrition, and predisposing to prostatic hyperæmic, or

it may result from orchitic atrophy, the result of a metastatic implication from mumps. It may be dependent upon some injury or operation upon one or both orchids or vasa deferentia. Moreover it is met with in spinal and cerebral lesions, either from damage or disease, or it complicates a case of traumatic neurasthenia. It occurs as a sequela of a debilitating influenzal attack, or as an incident with myxoedema. At other times it is the expression of a toxic state, either of a pathological or therapeutic nature, for instance, as in diabetes and albuminuria, or with plumbism. In some cases it seems to be conduced to or originates from excessive mental worry in relation to business or domestic affairs.

Impaired virile power may declare itself during any portion of what are usually considered to be the vigorous decades of life. It is subject to great variations in degree and duration, according to its etiology and the correction or remediability of the exciting factor. It may be temporary, relapsing, stationary, or progressive, eventually culminating in absolute and permanent impotency. The onset may be gradual or sudden according to the cause. The temporary condition may be partial or complete whilst in existence, the procreative desire being present only at distant intervals, and then of an evanescent nature, or coupled with incomplete or feeble priapism, or there may be a total absence for a prolonged period of all sexual feelings or manifestations. In some cases this would seem to be entirely dependent upon a neurotic or hysterical state. In others it is bred by a self-conscious fear of predestined failure. In both instances it is a mental inhibition of what subsequently prove to be perfectly functional organs. Partial impairment of a totally different nature occurs in cases of prostatitis in which there exists a chronic hyperæmia of the mucous membrane covering the veru montanum and the adjacent pocular sinuses of the ejaculatory ducts. With these, however, the psychological instinct is present and often increased to a pitch of great impetuosity, but owing to a hyperæsthetic state of the external generative organs combined with an abnormal sensibility and reaction of the lumbar centres to reflex stimulation, a premature climax results which is sometimes independent of or simultaneous with contact. Dissolution is followed by a marked sense of nervous prostration or bodily exhaustion which persists for many hours ;

consequently a considerable interval elapses before any attempt at repetition is desired or possible. Some patients experience prostatic discomfort as a sequela. It is either of the nature of burning, perinæal fulness, rectal tenderness or tenesmus, or of unpleasant, indefinite sensations in the suprapubic, inguinal, or lumbar regions. If the urethra be examined it invariably shows an excess of moisture. A diametrically opposite condition of impairment to the preceding is that of orgasmic failure. The psycho-vascular mechanism acts more or less perfectly up to a certain point and then fails, so that the cycle never, or rarely, proceeds to finality. This is due either to involuntary premature subsidence or to cessation of effort from muscular exhaustion combined with a lack of stimulus, owing to the blunted sensibility of the special sensory nerve-endings, so that they are anæsthetic to friction. Sometimes it is due to a faulty contraction of the ischio- or bulbo-cavernosus muscles, the latter of which should effectually and tonically compress the dorsal penile vein. Or it may be consequent upon an absence of the requisite secretions, either from defective vaso-motorial action or from degeneration or impaired vitality of the elaborating cells. There is often a marked mental element present in these cases which tends to intensify the condition. It is the consciousness of past failure which has arisen from a series of disappointments, and which engenders a recurring inhibitory sense of potential distrust. Paradoxical as it may sound from what has already been said of excess, impaired virility may follow a period of prolonged chastity in those who were formerly unaffected and capable. It is, however, usually of transient nature, and seems to be dependent upon physiological inertia from disuse, though it is sometimes conduced to by long usage of sedative drugs, combined with excessive tobacco smoking. Bromide of potash is the salt most commonly causative of the condition, but there are many other preparations which exert a spinal depressant or an aphrodisiac tendency. The bromides and iodides of potash, soda, or ammonia, carbonate of soda or nitrate of potash in big doses, camphor, belladonna, conium, gelseminum, stramonium, tobacco and salix nigra all possess this property to a greater or lesser degree. Alcoholic saturation, as in constant nipping, acts as a sexual depressor. In some cases it is a marked adjunct of the condition, if

not the principal cause. The direct bearing of the nervous system in the production of the state is noted in injuries to the head or spinal cord, in cerebral hæmorrhage, paraplegia and with ataxia, usually a parasyphilitic phenomenon. When impairment results as a sequence of grave lesions of the columns, tracts or centres, such as are prone to be of a progressive nature, the condition naturally tends to increase *pari passu* with the advancing implication and degeneration of the governing nervous elements, so that all efforts towards retardation or restoration are predestined to failure. But in certain cases where cellular or focal compression, such as is caused by extravasated fluid or by the products of inflammation, is relieved by absorption, then there may be a reaction of improvement. The maximum of stationary amelioration will depend upon the number of the undestroyed cells remaining and their individual vitality, or upon the intensity and freedom of passage of ascending and descending cerebro-spinal nervous currents. In traumatic neurasthenia, sometimes even with the slighter cases, the condition may be marked and persistent, so that the sexual function is totally in abeyance or highly impaired for many months, even when convalescence in all other respects is apparently established. In fact, some patients seem to think that they never regain that degree of virility which they enjoyed prior to the accident. Occasionally syphilis is responsible for impairment of sexual appetite and power through severe diffuse and persistent gummatous invasion of the testicles. The pressure exerted by an infiltrating and compressing fibrosis upon the essential spermatozoic cells may lead to their destruction and the subsequent atrophy of the organs, as is seen when the process undergoes resolution and absorption from appropriate treatment. The tissue destruction and the scars of repair that attend a more focal gummatous degeneration do not, however, seem to have a similar effect, even if they may have impaired fecundity through damage to the seminiferous tubules. The bulk of cases of syphilitic testicles experience no potential loss, it is the exception, not the rule, and is limited to only a small percentage of the diffuse infiltrations affecting both orchids. Dual castration is necessarily followed by impotency. A similar result supervenes on ligation or excision of portions of both vasa deferentia, consequent upon

testicular atrophy. But marked impairment of virility sometimes ensues after the removal of one orchid, notwithstanding that the solitary representative seems healthy and mature. I have seen a few cases of this nature. In one the left testicle was found to have necrosed a few days after a radical operation for varicocele, and was therefore removed. The patient subsequently married, only to find himself incapable, as he still was when last I saw him, of consummating one of his vows. The remaining organ was apparently a good specimen of a healthy and well-developed testicle, but nevertheless he was to all intents and purposes impotent. It is sometimes taught that a monorchid remains practically as potential and fecund as he was before mutilation. That some of these do beget children is unquestionable, but in all cases with which I am acquainted they have admitted that they lack the former power which they possessed when the organ was in duplicate, not only to the desire, but also as to its expression. The question naturally arises when only one organ has been ablated, what is likely to be the effect of the withdrawal of a moiety of the testicular secretion upon the body at large and the nervous system in particular, especially in relation to its sexual aspect? Judging from the feminine masculinity of eunuchs which presumably shows that the missing organs exercise not only a nutritive, but also a developmental function on the tissues in general, one would expect with a monorchid, even if the remaining testicle was perfectly healthy, that some special effect would result. I think one may premise that the physiological activity of the testicles is trophically governed by the nervous system, and that they are subject to the same vaso-motor law which presides over all secreting glands. It requires no great stretch of imagination to deduce that the orchitic secretion, like that of the thyroid body and other special organs, has in its turn some nutritive or stimulating action on the cerebro-spinal system, and that the deficiency of half of the manufacturing mechanism, and therefore its equivalent secretion, would be followed by some result. The following is a strong argument by analogy against this contention. It is well recognised in thyroidectomy that so long as you leave a sufficiency of the gland that myxœdema is not induced. In fact, the patient seems to get along as well with a portion as with the entire organ. Whether this is so, and

that the tissues are at their highest summit of vitality, it is impossible to say. Certainly the grosser manifestations of the disease are absent after partial excision of the gland. Impaired virility, even to the degree of impotency, is an incident with myxoedema, unless the disease be efficiently controlled by compensatory thyroid feeding. The effects of this perverted nutrition are probably of a dual character, partly local or testicular, but mainly cerebral. It is as one would expect, considering the mental state of a myxoedemic case, as evidenced by the retarded speech, the slow evolution of thought or movement, and the general bodily condition of mucigenous deterioration. When impairment arises in connection with diabetes or albuminuria one has the direct provocative effect of the glycosuric and uræmic state, respectively, acting on the central nervous system or else general debility from malnutrition, due either to excretory loss or tissue toxæmia.

Phosphaturia is often troublesome when a marked functional element pervades a case. This excess of phosphates is apt to alternate with an increased excretion of either oxalates or urates. It results partly from the excessive mental anxiety usual to those afflicted, but it is chiefly due to faulty assimilation of food and to imperfect metabolism or oxidisation of waste products. When an hyperæmic condition of the prostate or urethra exists, the frequent and profuse passage of phosphatic crystals, acting as a mechanical irritant upon the hyperæsthetic mucous membrane, causes discomfort, and thus serves to intensify the existing hypochondriacal state. With these cases the symptomatic neuroses are usually in evidence, such as uncontrollable blushing, undue palmar perspiration, vertical headache, faucial globus, and so forth.

It will be seen from the foregoing remarks that there is scope in these cases for discrimination, not only as to the existing state, but also as to the exciting cause. Whatever knowledge the bulk of medical men have of this subject is usually gleaned by personal observation on their own patients rather than from monographs or text-books. The literature bearing upon it is not only limited, but such as exists is scanty, and avoids entering into necessarily unpalatable details, so essential for differential diagnosis and appropriate treatment. This feeling is also apt to unfavourably influence the unravelling of a case. The requisite interrogation of the patient

is an unpleasant matter, so that a brief and constrained consultation is prone to result and to be concluded without the *fons et origo mali* having been discovered. I think that this reserve of cross-examination, which the patient construes as want of interest in his case, is one of the reasons why so many of these sufferers drift into the hands of predatory quacks. These gentry not only work upon their hopes and fears, but shower upon them a plethora of ignorant attention, feigned sympathy, and repeated promises of eventual cure, that is, so long as the usual financial consideration is forthcoming. Needless to say, the assumption or admission of the existence, quality, and degree of a state of impaired virility, often depends solely upon the patient's statement to that effect, all probable or presumptive causative or corroborative signs being absent, unless, as in the case of a married man, confirmatory testimony can be obtained from the wife. As regards the latter evidence it must not be forgotten that the plea is occasionally used by husbands to cloak a diminution of former marital intercourse owing to the bestowal of favours elsewhere. On the other hand there may be irrefutable history or signs of former injury, the past or active indications of definite disease such as is usually associated with sexual impairment, so that it would be highly improbable that the function should have escaped deterioration. In forming an etiological diagnosis, one must primarily settle that one is really dealing with an acquired and not a congenital phenomenon. Secondly, as to whether it be due partly or entirely to purely local conditions, and not dependent chiefly, if not absolutely, upon the nervous system, or that it is not simply a symptom of some constitutional disease. Should a nervous origin be apparent then one must determine as to its functional or organic nature, and in the case of the latter as to whether it be a cerebral or a spinal lesion or a combination of both with a predominance of one of them. As regards the customary local or genital causes, these are detected by visual and digital examination. Having decided as to the etiology of a case, another question then arises. Is it amenable to treatment, and if so, upon what lines should this be conducted? It must be self-evident that when one is dealing with progressive nervous disorders, or hopelessly degenerated cells and structures, that the answer must be in the negative. A similar

non possumus holds good with injuries that have resulted in section or laceration of the spinal cord, in destructive brain lesions, or after removal of the testicles. With a few organic neuroses, however, there may be some hope of improvement, notwithstanding the usual primary gloomy outlook, such as in paraplegia, where pressure might be surgically relieved or which may react to anti-syphilitic remedies, or in the early stages of certain ataxic cases, and, as sometimes happens, in transverse myelitis, after subsidence of the acute inflammatory stage. As regards the possibility of improving impaired virility in locomotor ataxy, this can only be hoped for whilst the disease is, so to speak, in its infancy, and, perchance, capable of being favourably influenced by treatment. I am aware that there are strong views against regarding and treating ataxia as an active syphilitic rather than a parasymphilitic state. It is asserted that such a line of medication does not benefit it, but in fact often tends to make it worse. With all deference to this theory, true as it is of advanced cases, nevertheless, in the early stages of tabes that has supervened upon a comparatively recent syphilitic infection, a patient may regain the lost function, and even prove fecund, if only temporarily, after a judicious course of mercurial inunction, when other methods, such as organotherapy, suspension, etc., have failed. Personally, I have never been convinced of a beneficial result following the administration or injection of spermin in any variety of sexual deterioration. Perhaps my experience is singular, but I have given it a fair, impartial, even if unsuccessful trial in all varieties of the condition. In the organic nervous cases our resources are decidedly restricted. They practically consist of hygienic and dietetic measures, combined with the administration of nervine tonics, unless some special reason contraindicates their exhibition, such as the exacerbation of the ataxic lightning pains following strychnia. The most commonly prescribed stimulants are strychnine or its derivative nux vomica, some preparation of arsenic, quinine, phosphorus, or one of the numerous salts of iron; but where a strong functional element exists direct benefit may be obtained by primarily employing an antispasmodic, such as asafetida ammoniacum, valerian, valerianate of zinc, or one of the bromides. This method may prove successful

by itself, but it is usually necessary later on to resort to nervine stimulants or aphrodisiacs. These are best exhibited as soon as the hysterical state has shown definite signs of abatement. The following drugs possess and exercise a tonic property: strychnia, damiana, arsenic, phosphorus, cantharides, cannabis indica, yohimbine, opium in small, and iron in large, doses. The tincture of perchloride of iron in full and increasing dosage seems to be the most potent of the ferruginous series. In cases resulting from traumatic neurasthenia it may be advantageous to employ the Weir-Mitchell treatment. When an exceedingly hyperæsthetic state of the generative organs causes impairment by precipitation it will be found that bromide, with or without ergotine, or salix nigra are useful. These cases, however, frequently require topical treatment to the prostate, either by instillation or painting with solutions of nitrate of silver. In some cases the irritability may be improved by the use of rectal suppositories containing hamamelidin, belladonna, and morphia, or by the employment of ice-cold enemata. When orgasmic failure seems to be due to a loss of sensibility of the nerve-endings in the glans penis the application to the mucous membrane of some liquid extract of echinaceæ augustifoliæ by means of a camel's-hair brush may produce the desired effect. In some cases rest, with change of climate, scene, and surroundings, as in a sea voyage, may result in rehabilitation. Should a large varicocele be the presumptive contributory cause of the impairment it would furnish an additional factor of consideration for advising a radical cure. In several cases where I have removed a portion of the varicose pampiniform plexus for the usual surgical reasons the patients have subsequently volunteered the statement that their sexual powers have been markedly improved after the operation, and that they have benefited considerably in a manner they were not led to expect.

I have been much disappointed with the small amount of benefit that results from the employment of electricity, even in instances where one might have expected it to effect great improvement. But it is a legitimate form of adjunctive treatment in functional and neurasthenic cases, and, notwithstanding the ill repute that it has gained from being exploited by the unqualified parasites of our profession, it should be borne in

mind and utilised in suitable cases. I look upon it rather in the light of a placebo, and as a means of saving the sufferer from falling into the clutches of those charlatans who endeavour to persuade them that it is the panacea for all virile and genital defects. Your patient is sure to hear of its marvellous effects, and if you do not prescribe its employment and convince him of its overvaunted results, well, he will probably drift to the sharks.

A

PLEA FOR CÆSAREAN SECTION.

A Lecture delivered at the Medical Graduates' College and Polyclinic, June 26th, 1901.

By Professor W. JAPP SINCLAIR, M.D.

(Concluded from p. 38.)

4. *Should the uterus be drawn forward or cut in situ?* I would recommend an inexperienced operator to make the wound sufficiently large to enable him to draw forward the uterus out of the abdominal cavity. It is the safest proceeding, and it is the one which is invariably followed by those operators who make a transverse incision in the fundus uteri. I do not think there is much difference in the results obtained by experienced operators, whether or not they "eventrate," but I can well understand that without very efficient assistance, the operator who incises *in situ* will usually find that a considerable quantity of blood and amniotic fluid has escaped into the abdominal cavity. It is little comfort to be assured that these fluids are not septic. Neither is healthy urine.

5. *As to the incision in the uterus.* There has been a curious discussion here again. It is strangely analagous to the ancient controversy on the site of the abdominal wound. The plain man who had to do the operation for the first time would naturally think that he ought to make the incision in the middle line, just opposite the incision in the abdomen. He might find the uterus lying a little oblique, and require to have it steadied by an assistant in order that he might make the wound central and symmetrical. That is all obvious

enough. But operators on the Continent of Europe have "sought out many inventions." Fritsch introduced a transverse incision in the fundus, and there now is a regular school, mostly composed of young operators, who strongly advocate the "Fundusschnitt nach Fritsch." Johannowski makes a posterior sagittal incision; Müller prefers an anterior sagittal incision running up to the fundus; Caruso has a modification of Fritsch's fundal incision, but in what respect it is not identical I cannot make out from the description; and Kehrer of Heidelberg recommends a transverse wound situated anteriorly in the lower uterine segment. After all, the incision in the middle line and in the middle third, or as nearly so as practicable, appears to be the easiest and best. The proposed modifications are not necessary to immediate success; some of them may even be detrimental. In seriously defending them the seniors,—the masters,—do humanity a disservice, for the juniors immediately make them matters of principle, and they fight duels, that is to say, engage in logomachies, in defence of the *verba magistri*, making in their zeal much of their medical literature unprofitable and even unreadable. But for the seriously deterring effect which the multiplication of small details as if they were important steps in the operation has upon conscientious beginners, these differences about trifles might be set aside as but the levities of grave men. The essential thing is to make a clean wound, and let it be absolutely aseptic and sufficiently large for the purpose. It is much better to have it an inch too long than a trifle too short; to have it long enough to permit of the prompt extraction of the fœtus, than to have either to enlarge it or to tear the uterine tissues.

6. *The Prevention of Hæmorrhage.*—The alleged danger from atony of the uterus hardly exists. There may be a distinct difficulty in getting out the membranes, but there is usually no difficulty in stopping the hæmorrhage. As soon as the secundines are removed the uterus contracts and the hæmorrhage ceases. In spite of all that is said to the contrary I should recommend an inexperienced operator, as Leopold of Dresden does, to place an elastic tube around the neck of the uterus, and have an assistant ready to draw it tight if there is bleeding. This appears to me to be a safer and more convenient practice than entrusting the broad ligament to an assistant, who must put his hands

into the wound in order to exercise efficient control. Under the elastic band the uterus remains flabby during the suturing, but in every case of mine I have watched the blood returning when the ligature was gradually relaxed, and as soon as the bright red colour of the uterus was restored there was a resumption of the contractions. If there is anything approaching atony a little manipulation through an aseptic moist cloth wrapped over the uterus makes it resume its condition of a firm spheroidal mass, such as we feel in an ordinary case after delivery. Leopold had to remove the uterus in five cases owing to atony alone, out of seventy-one intended to be conservative, and R. v. Braun-Fernwald had to remove it on account of hæmorrhage from the suture canals. From my own observation I cannot but think that kneading of the uterus after the suturing is complete would make such a serious addition to the operation unnecessary.

7. Another question which has received some attention: *Is there any use for ergot of rye* in these cases? I have had ergotin injected two or three times soon after operation, but it seemed to cause intense pain. The effect has been in my opinion so markedly unfavourable that I would not allow ergotin to be administered again to a patient of mine, unless I was satisfied of the existence of atony with some considerable hæmorrhage. But I have not seen anything to give rise to the slightest anxiety. There may be some use for ergotin employed as a uterine tonic before operation, as recommended by R. v. Braun-Fernwald, but of this I have no experience in connection with Cæsarean section.

8. Then there has been much discussion about the *sutures*. In my opinion the suturing should be exact. I have invariably used fine silk for the deep sutures and catgut for the superficial, and I have inserted as many as twenty of each kind. They should be drawn pretty tight, to make allowance for the involution of the uterus. Inclusion or avoidance of the decidua is, I believe, a matter of no importance. Each proceeding has its advantages and its drawbacks, but neither is among the essentials.

With regard to the operation as a whole, it is probably the most easy of all the abdominal sections. If this be true, why should not Cæsarean section be done more frequently in suitable cases, instead of perpetuating the practice of destroying a

living child, which in thirty seconds might be given a separate existence? Is it because, though easy, it is the most dangerous of all the operations on women? By no means. We are mere slaves of the tradition of danger. We frequently do more dangerous operations with a light heart and with an easy or a sleeping conscience, and without the justification. In many of our meetings of gynæcologists, at home and abroad, surgeons young and elderly, inexperienced and experienced alike, bring for exhibition tray-loads of fibromyomata and other tumours of the uterus of all shapes and sizes, which they have obtained by abdominal or vaginal section, by myotomy, myomectomy, hysterectomy, panhysterectomy. The fashion has changed; the trays were wont to contain tubes and ovaries. But a man cannot nowadays acquire a reputation as an abdominal section gynæcologist if he does not bring trophies of his enterprise and skill to the notice of admiring or envious *confrères*. It is the custom, and he must conform or feel that he is doomed to a position of obscurity and inferiority. And if there are not sufficiently numerous and curious tumours to show, or if the population of the place of meeting cannot provide clinical material for exhibition operations, we get up magic-lantern entertainments to indicate by means of living pictures how the specimens are obtained, and we delude ourselves with the notion that we are assisting at a scientific *séance*, instead of at a grotesque, coarse, and misleading spectacle. Now every-one of the operations by which these specimens are obtained is as dangerous to the patient as Cæsarean section need be. For instance, abdominal hysterectomy, when it is a legitimate operation, as is sometimes the case, is usually a more serious operation than Cæsarean section duly arranged for. You may ask, Would I say, then, that Cæsarean section ought to be undertaken outside the hospitals, and by any practitioner who meets with absolute indications in a case? By no means *as yet*, but I believe the time is coming when that will be the recognised practice; not, however, until the profession take more scientific interest in midwifery, and the public have acquired greater confidence. There is a large reserve of cases in which it could be done with comparative safety now, and I am glad to see reports appearing now and again of cases in which the operation has been successfully performed in country districts and in small towns in England;

and probably there are other such cases which have not yet been reported. The cases which I would exclude as outside the range of practice of the inexperienced and isolated operator in the meantime, are chiefly those emergencies when he is taken unprepared, which is not, of course, the case when abdominal hysterectomy for tumour is contemplated. The category of cases which should be, as a rule, excluded as yet from general practice are those for carcinoma of the uterus in the parturient, for they are too dangerous. The mortality from sepsis by bacterial invasion under the most favourable conditions is as yet very high, and even success is almost occasion for regret. Cases of eclampsia should also be excluded in the meantime. If you take a review of the work of the world for ten years you will see that the mortality after Cæsarean section in cases of eclampsia is very great, but it is impossible to say to what extent death was to be attributed to the condition which produced the eclampsia or to the operation. Placenta prævia in certain cases is one of the indications which will have to be considered sooner or later. The late Mr. Lawson Tait strongly urged the adoption of Porro's operation on account of placenta prævia, and it must be admitted that under conditions there is much to be said in support of the proposal. He was probably only premature. What used to be lauded, and is now extensively practised, as the best method of treatment in placenta prævia, was the German method of turning the child, bringing down a leg or hip, and leaving the rest to spontaneous expulsion by the uterus. That method has greatly diminished the maternal mortality, but it results in the almost certain sacrifice of the child's life. Craniotomy does not claim so very many more victims. I should also exclude parturition complicated by tumours which have remained concealed so long that proper arrangements cannot be made until the uterus must be exhausted, and when the appliances for Porro's operation are not within reach. In the same way we must set aside rupture of the uterus. If the uterus is allowed to rupture, or if futile efforts have been made at version or craniotomy, or when the patient herself is a sufferer from some general organic disease, which is likely sooner or later to be fatal, our general practice for yet a while must remain unchanged. But after setting aside all these classes, many cases still remain in which Cæsarean section is known

to be indicated. Among my fourteen cases the indications have been produced by hip-joint disease, extreme deformity from rickets, injury to the lower extremity in infancy, infantile paralysis, lumbar curvature, and even osteomalacia.

Osteomalacia has been practically exterminated by social legislation, and is now about as rare as leprosy in this country. But too many causes of extreme pelvic deformities still remain. There is a great reserve of cases in the country on which the operation of Cæsarean section might be done under the most favourable conditions. Just as in the history of ovariectomy, when it was shown to be a comparatively safe operation large numbers of cases came under the care and skill of the surgeon which had hitherto been considered by the medical practitioner too advanced or complicated for operation. So again, when vesico-vaginal fistula was found not to present a hopeless condition, and not to be absolutely incurable, a vast reserve of cases began to be drawn upon by the operators, which kept them constantly employed for the greater part of a generation. Analogously, there are living amongst us at the present time a large number of women who cannot by the adoption of any measures known to the obstetric art be enabled to bear a living child, however frequently they may become pregnant. If you take, for example, my tenth case, you will find a fairly typical history. The patient was pregnant at full term for the sixth time. Every previous child was destroyed by craniotomy or by the induction of labour too early for genuine viability, and craniotomy had to be resorted to in the case of twins, and even after induction of labour at seven months. Such cases present a very embarrassing ethical question,—“On what criteria are we to arrive at a decision whether to operate or to refuse to operate in any such case?” It was comparatively easy to decide in my thirteenth case. On a former occasion the patient had been warned and advised with regard to induction, and she promised if she became pregnant again to come to the Maternity Hospital before the end of the seventh month; but she did not put in an appearance till close on full time. The delay was intentional. She was now at the end of her ninth pregnancy. The explanation of the apparent negligence, as given to the head nurse in the hospital, was that she did not wish to have a living child; her husband, in fact, refused to have

a child born alive as he "did not want to be bothered with it." So here was a revelation; this woman had gone to the Maternity Hospital and elsewhere time after time expecting to be accommodated by the murder of her children within a few hours of the time when but for her well-known deformity they would have acquired civil rights. The situation appeared to me altogether monstrous; and I refused to kill the child. The woman was not in labour, and she had the option of applying at another institution. She decided in favour of Cæsarean section; the time selected was a few days before the onset of labour; the operation was performed, and the patient went home with a living child. These cases belong to the class to which I desire to call special attention now. They are easily recognised; the diagnosis is clear from the obstetric history alone; no pelvimetry is necessary. The time of operation can be arranged; the surgical proceedings are easy; and there is a comparative absence of danger. If we are justified in repeatedly performing craniotomy, or in bringing on early premature labour in such cases, would it not be more honest and dignified conduct on the part of the accoucheur, and less dangerous to the patient, if abortion were brought on in an early month of pregnancy when "facultative" sterility could not be ensured? If such amiability be not mere cant or hypocrisy or self-deception on the part of the doctor, it can hardly be ranked as scientific obstetrics when practiced by an experienced specialist. Yet in the course of a discussion on this subject only quite recently, an experienced obstetrician stated that he had just done craniotomy for the tenth time on the same woman, knowing it to be the tenth time. One would prefer to believe that such incidents are now mere exceptions, for we do not all accept the German dictum that the wish of the pregnant woman must be decisive. Professor M. Cameron operated on a woman in her twelfth pregnancy; she had gone to full time in her eleven pregnancies in very rapid succession—ten of them in thirteen years,—and every time embryotomy had been done in conformity with her wishes; Cameron refused to oblige her and insisted upon Cæsarean section, and he operated successfully.

There is no use in discussing the question of repeated abortions or embryotomies on formal ethical grounds; we have no Court of Ethics

for whose sanction we can appeal in this matter.

Pinard, of Paris, the great advocate for the preservation of foetal life, argues that the physician must be the arbiter. Frenchmen may have a racial standard of ethics and a special point of view, but it is safe to predict that as far as the world in general is concerned Pinard will assert such claims in vain. The doctor is only the arbiter of his own actions; he is the keeper of his own conscience. If he thinks it is a crime to perforate the head of a living child at full term, or to kill it under the pretence of inducing premature labour, he can refuse to be *particeps criminis*. It is vain to lay down original ethical dogmata for the control of other men's actions. It is only by the formation of a professional opinion sanctioned by success, and then by the evolution of a public opinion, that we shall get those cases of known contracted pelvis properly treated, and Cæsarean section will become in certain categories of cases the recognised and ordinary operation. And we are making some progress. Barnes wrote over fifteen years ago:—"If the operation could be done at a chosen moment, and so improved as greatly to increase the probability of saving the mother, then the already high probability of rescuing the child might turn the scale in favour of the Cæsarean section, against embryotomy."

Writing ten years later Säger could say: "The medical practitioner who does craniotomy on the living child in a case in which the patient could have been removed to a hospital in order to undergo Cæsarean section with preservation of the child's life, has fallen behind the requirements of the times."

In conclusion, I should like to call attention for a moment to another class of case—that which must be operated on in emergency. Such cases take the medical practitioner by surprise, and he must do the best he can with the means at his disposal. With regard to these cases I should like to urge most strenuously the need for prompt diagnosis and decision in action. Cæsarean section should not be considered as a last resort after futile efforts at forceps delivery, version, and even craniotomy have been made. It is easy enough to spoil a case for Cæsarean section by vacillation and by injudicious attempts at treatment. Usually the cases are obvious enough. But with regard to cases which

are doubtful, such as in deformed primigravidæ, examinations should be reduced to the minimum and cannot be made with too great care. Veit, of Leyden, considers it essential to success that no vaginal exploration should be made within three weeks of the time at which the Cæsarean operation is to be performed; any vaginal examination nearer to the time contra-indicates operation. In the present state of our knowledge we may without offence consider Veit's precept a counsel of perfection. I merely mention the opinion to indicate the conviction of thoughtful and responsible teachers of obstetrics that absolute asepsis of the genitals is essential to success. The extreme of unconsciousness of such requirements is illustrated on the other hand by a case mentioned by one of our British operators. He was asked to perform Cæsarean section in a case in which two or three medical men had tried all the other known methods of delivery in dystocia, including craniotomy. After two days' effort, which resulted in perforating the bladder and opening Douglas's space, they proposed that Cæsarean section should be resorted to. R. v. Braun-Fernwald refers to a case in which forceps were first tried, but the instrument slipped and lacerated the perineum. The accoucheurs then tried version, and failed. So they resorted to Cæsarean section, and extracted only an anencephalic monster after all.

Such facts and opinions might be quoted to an indefinite extent, but the lesson taught by them must be already plain. It amounts to this, that in the difficult emergency cases we should endeavour not to compromise the patient's position by aimless, ineffective, and frequently repeated examinations, but according to our knowledge, our judgment, and our opportunities make a diagnosis promptly, and take decided and consistent action. Shillyshally is as ruinous in obstetrics as it is in politics. Neither men nor Nature will tolerate it.

In the foregoing pages I have endeavoured to show by the historical argument that we are still under the influence of the dread of the Cæsarean operation produced by its traditional mortality, and by the spectacles of human suffering conjured up by the recitals of the histories of individual cases in the hands of the old obstetric surgeons. We have not yet adjusted our views of the operation in accordance with the everyday occurrences of modern abdominal surgery.

To emphasise this fact by way of contrast, I have called attention to the extreme degree of freedom which we permit ourselves in resorting to abdominal operations for conditions which do not invariably demand operative interference; while the operations performed have usually an inherent danger as great, if not greater, than Cæsarean section for absolute indications, and apart from emergency circumstances.

In differentiating the various classes of cases, one stands out distinct from all the others, to wit, that of the women who are well known to be the subject of deformity of the pelvis inconsistent with the possibility of maternity. This is the class of case in which due preparation can be made for operation as for ovariectomy. The present routine practice with regard to these cases consists in early induction or embryotomy. An endeavour is here made to show that in such cases Cæsarean section may be performed with comparative freedom from danger.

It is further suggested that with expanding indications the other categories will come under consideration as to their suitability for emergency operations in general practice; but the time for general acceptance of them as such has not yet arrived.

Lest I should be supposed to treat the subject with levity, or to recommend reckless and ill-considered operating, I may best express the spirit of my advocacy by finally quoting the words of von Winckel, written thirteen years ago, "There is still a mortality of 8.6 per cent. after Cæsarean section (this was recently 54 per cent. according to Meyer—of 1605 women 867 died) even by the most experienced operators; half of these were cases of infection. I am of the same opinion as Wyder and J. Veit, that the old mortality will soon be reached if the operation is undertaken very frequently outside of well-conducted institutions;" which appears to me to amount to this; that the operation should not be performed unless everything which available knowledge, foresight, and experience can accomplish in bringing the surgical and obstetric treatment up to the "hospital" standard has been employed in order to eliminate the causes of failure.

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A CLINICAL LECTURE ON FRACTURE OF THE SPINE.

Delivered at St. George's Hospital, March 19th, 1901.

By CLINTON T. DENT, M.C., F.R.C.S.

GENTLEMEN,—There are two cases in the hospital at the present time under my care, of fracture of the spine. It is to a few points in connection with fracture of the spine generally, and especially with regard to the possibilities of operative treatment of these cases, that I want to direct your attention. One of the patients has been in the hospital for some eight months. The second patient was admitted only a few days ago. The injury in both cases was very similar, and affected the upper dorsal region. From the clinical point of view, the cases, while strongly contrasted, supplement each other very effectively.

In the case which has been here now for some eight months in the hospital, the symptoms were acute at the outset, and they varied from day to day, just like those of the man who was more recently admitted. Gradually, however, the acute symptoms subsided, and now, for some months past, there has been scarcely any change to note. There has been no improvement, but the man seems no nearer his end than he was six months ago. The case, therefore, is instructive as illustrating the extent of recovery that is possible in fracture of the spine with grave injury to the cord in the mid-dorsal region.

The second patient, however, to whose case I desire more especially to draw your attention, never passed out of the acute stage. A rapid succession of changes occurred almost from day to day. Here, again, the injury is in the dorsal region, but to the best of my belief a little higher up than in the chronic case; in one the injury, as regards the spinal cord, appears to be about the level of the sixth dorsal vertebra; whereas in the other, it would seem to be the level of the second or third.

In the case of the second patient, the acute case, the history of the accident could not be fairly ascertained. All we know is that the man, who is a stableman, fell from a hayloft on to pavement or cobble-stones—a height of some ten or twelve feet. He clearly struck his head in the fall, for there was a scalp wound, and he was also, on admission, suffering from cerebral concussion. There were no marks or bruises that enabled us to form an opinion as to the part of the spine that was injured. There was no irregularity of the spinous processes. The case was therefore a rather complicated one, and it was not very easy to disentangle the symptoms and to determine at first what symptoms were due to the head injury and what to the spine. Without entering minutely into his symptoms, I may say that I came to the conclusion that there was a pretty severe concussion of the brain, and also evidence of a tolerably diffused injury of the spine, as well as of a grave lesion at about the level of the second or third dorsal vertebra. Before very long consciousness returned, accompanied by vomiting, as one would expect in a case of concussion of the brain. It was now abundantly clear that the worst of the symptoms were due to the spinal injury, for there was complete paralysis of the lower extremities, and the breathing was largely diaphragmatic. The vomiting was very severe and persistent. This may have been partly due to the brain injury, but in cases where the upper part of the spinal cord is injured, whether by fracture of the spinal column or by extravasation of blood, or by simple concussion, persistent vomiting, lasting often for days or weeks, is frequently a marked feature of the case. Mr. Erichsen records an instance where the vomiting continued for a great length of time. I can recall, too, a case—that of a young gentleman—who was thrown out of a runaway carriage, and who had in addition to a compound depressed fracture of the skull evidence of injury to the upper part of the spine. Here the vomiting was extremely persistent, and went on for some three weeks after the accident. I look, therefore, on persistent vomiting as indicative rather of mischief in the upper portion of the cord than in the brain. In the compass of a lecture I can do no more than allude briefly to a few of the more noticeable symptoms and changes

that were present in these cases, and it is best, perhaps, to deal with them not in the order so much in which they were noticed, but rather as they affected the different physiological functions. First with regard to the respiration. The movements of respiration in both cases were to a great extent, but not entirely, diaphragmatic. You can easily deceive yourselves as to the extent to which the abdominal or thoracic muscles really work in these cases. As we watched the breathing the lower part of the abdomen was seen to protude at each inspiration, very much as it does in a rickety child in whom the thorax is too soft to admit of the proper expansion of the chest walls. The abdominal muscles were wholly paralysed, but the elastic recoil of the belly, following the thrusting forward of the abdominal wall by the descent of the diaphragm, gave the impression that the muscles actually contracted. So, again, the thoracic muscles appeared to be doing more work than was actually the case. But the thoracic muscles of course were not wholly paralysed. Supposing, as we did suppose, from the evidence derived from the loss of sensation, that the main injury to the cord was about the level of the third dorsal vertebra, the phrenic nerves, of course coming off well above the site of injury, were working perfectly well, but the posterior thoracic—Bell's—external respiratory nerves were also unimpaired. So, again, some of the other nerves, which could assist in the work of respiration, either by acting upon accessory muscles of respiration, or upon the ordinary muscles of the upper part of the chest, came off above the site of injury. In such cases it usually takes some time and study of the symptoms to work out accurately the site of injury as evidenced by paralysis of movement or sensation, and it is well not to judge too hastily, for the patients' accounts of their own sensations, or loss of sensation, are by no means to be relied upon. Repeated investigation is necessary in order to make certain of the diagnosis. A rough, but still on the whole a sufficiently accurate, guide to the segment of the cord from which the various nerves arise is the following:—Assume that the nerves arise from the cord (this is of course a different level from their exit from the intervertebral foramina) at equidistant points throughout. We may consider that the cervical nerves start from the cord, at

equal distances, between the occiput and the sixth cervical spine. So, again, the upper six dorsal nerves may be assumed to come off at perfectly regular intervals between the sixth cervical spine and the fourth dorsal spine, and the lower six dorsal nerves at equidistant points in the segment of the cord between the fourth and eleventh dorsal spine. This easily remembered scheme will be found useful when you first have to deal with cases of spinal injury. Some time must elapse before the exact site and extent of the cord lesion can be precisely localised. If the breathing of the acute case was closely observed, a peculiarity of the respiratory movements was very evident. As the phenomenon might easily deceive, it is important to notice it. When the hand was placed on the "accessory" muscles of respiration, attached to the head above and to the clavicle and first rib below, it could easily be felt that they contracted unduly at every inspiration. The lower half of the thorax moved with the respiration, and appeared also to be working, but close inspection showed that during inspiration, though the lower part of the thorax indeed moved, so far from expanding it tended really to fall in, owing, of course, to the paralysis of the intercostals at this level. The patient was a man of thirty-five, with a tolerably rigid thorax. In a younger person the falling in of the lower part of the chest wall would have been still more evident. On the other hand, the upper part of the thorax was doing increased work. The patient then breathed mainly with his diaphragm as a child would, and with the upper part of his chest as a woman does. He showed in fact diaphragmatic and what is called the superior costal type of respiration.

So far as the respiration is concerned there seemed no reason, therefore, why he should not live for a long time. Patients with almost purely diaphragmatic breathing die pretty soon as a rule; but it is very remarkable that in certain cases, even with fracture very high up in the spine, the diaphragm almost alone can suffice for respiratory purposes for a long time, in one case that I know of for many months. The compensatory nutrition of the muscle under the strain of extra work is a marvellous instance of adaptability. Another favourable feature in the respiration is that some of the muscles of expiration, though only of the

thoracic walls, can act. The man is therefore able to cough and to expel the mucus, which from the very first in these cases tends to collect abnormally in the bronchi. For the first two or three days the case looked very serious indeed, and there was a fear that the man would die practically of suffocation, owing to the difficulty of expelling the tenacious secretion of the bronchi. But then there came one of those sudden changes which are so characteristic of this class of injuries, and which a very short experience will teach you renders it very injudicious to give too precise a prognosis. Some four days after the injury the muscles had adjusted themselves, the rate of respiration was decreased, the mucous secretion had diminished, and the lungs were working throughout fairly well.

Next as to the circulation, under which I include the vaso-motor condition. Usually in these cases the pulse varies very greatly from time to time, as you would naturally expect from the pulmonary condition. The pulse rate varies greatly too according to the level at which the cord is injured. This patient showed some very marked vaso-motor disturbances, and to my mind the case furnished good evidence that it is incorrect to assume that there is a single vaso-motor "centre" in the cord. Regarding these cases clinically there seems to be not one but many points in the extent of the cord, all of which can exert strong vaso-motor influences. A day or two after the patient's admission, on examining his legs I found that the lower parts were stone cold; the skin was mottled and white like marble. It looked as if gangrene or, at any rate, sloughing to a considerable extent was impending. Bichat, as you know, classified the modes of dying as three, and spoke of death beginning at the head or the heart, or in the lungs. Here it seemed that death was beginning in the extremities. But on the next day, on looking at the part where we expected already to see serious changes indicative of rapid sloughing, we found that the skin was much warmer than the surrounding parts, indeed, the superficial temperature of the parts that twenty-four hours previously had seemed cold and dead was now well above the normal. Again one of these sudden changes. The circulation had entirely returned, and, yet more, the small vessels must obviously have all been dilated. There was not

only then a profound vaso-motor change, but also an extremely sudden one. The precautions for maintaining the temperature, local and general, had remained the same throughout. The temperature of the limbs underwent no marked change during the remainder of the illness, and the nutrition of the parts seemed fairly well ensured; but a day or two later a copious erythematous rash appeared very suddenly about the arms and chest, and the patient had bright hectic-like patches on the cheeks. This rash, which could not be accounted for by mechanical or local condition, was no doubt due again to the vaso motor disturbances. It disappeared completely, and almost as rapidly as it had appeared. These vaso-motor disturbances are not of any great gravity, but in their suddenness of onset and capricious disappearance they resemble the more grave symptoms that occur with regard to respiration and the digestive and urinary tracts, and the lesson to be drawn from them is—and it is one that experience of a very few cases of fracture of the spine will bring home to you—that while in these cases your prognosis must always be grave, it is always better to be very guarded in expressing it. These sudden fluctuations in the patient's condition may extend over a long period. Grave symptoms may arise and be recovered from on many occasions. On the other hand, when all seems going well your patient may suddenly run rapidly down hill and die in a few hours.

I can only allude now to one other evidence of vaso-motor disturbance which was well shown in this man, that is the symptom of priapism. This condition is common in injuries occurring high up in the cord, that is to say, the lower part of the cervical and the upper portion of the dorsal cord. Irritation much lower down may also produce the symptom. In total crushing of the cord the condition would probably be absent; in injury to the lumbar cord it is never present. Text-books on physiology, and on surgery too for that matter, are sometimes rather misleading on this point. It used to be taught in the physiological class, that there is a centre situated in the lumbar cord which on being irritated produces priapism, owing to the influence carried by the "nervi erigentes," as they are called. These are inhibitory nerves which bring about relaxation and, consequently, dilatation of the blood-vessels concerned. But you must

remember that this inhibitory influence can also descend from the cerebral centres. The priapism is very seldom persistent, and does not usually last more than a day or two, as in this case.

In rarer instances it persists for a long time, and it always disappears at death. The importance of the condition is, that it may add very greatly to the difficulty—always considerable—of combating the troubles that arise from the condition of the bladder. From the very first in these cases the urine has to be drawn off. We have to deal with an insensitive urethra and an insensitive bladder. The sensations of the patient furnish no help. Further, owing to the vaso-motor changes, the mucous membrane throughout becomes swollen. At first soft and easily excoriated or even lacerated, in a very short time it may become perfectly rotten. Even when the utmost skill and gentleness are exercised and the softest instruments are employed, it is very easy to tear the mucous membrane of the urethra, to make false passages, or even to inflict very serious injury on the bladder itself. A catheter should always be passed very slowly in all cases. Indeed, as a French surgeon well remarked, a catheter should not be "passed." The urethra should be allowed to "swallow" the instrument. In these special cases the more deliberate you are the less chance is there of inflicting any injury. An accidental injury is not the less a calamity of surgery because it is so easily brought about. A slight tear of the mucous lining of the urethra is a very grievous thing in these cases. You cannot allow time for the little tear to heal up by rest. The water has to be drawn off regularly, and once the slightest injury has been inflicted, difficulties set in which are sure to increase every time the bladder has to be emptied. The late Mr. Durham, in a case of fracture of the spine in which priapism was very persistent and troublesome, suggested an operation which I think might often be repeated with advantage. He performed perineal section in order to save the patient from the trouble and the hæmorrhage that attended the use of the catheter. In his case the operation was performed because it was found impossible to get a catheter into the bladder at all. The advantage of a large opening in the perineum would be that the bladder might be dealt with locally and freely without risk. It can be washed out rapidly with ease, and it must be remembered that serious bladder changes very

easily ensue in these cases. The incontinence of urine which would result from perineal section would not be more difficult to deal with than that which flows along the ordinary channel.

You may remember that I proposed to perform the same operation on this patient, but the man refused to have anything done. I suggested operation, however, in order to relieve a remarkable condition of the bladder which is not often likely to be met with in so marked a form. A few days after the man's admission he had very marked hæmaturia. This, we had reason to suppose, was quite independent of the use of the catheter; in all probability the bleeding was due to trophic changes extending along the greater part of the urinary tract, though, no doubt, largely from the bladder itself. The difficulty of passing the rubber catheter increased every day; the obstruction to its passage was situated either at the very neck of the bladder, or just within that viscus, much further back, therefore, than the difficulty is ordinarily met with. The urine got horribly offensive; the last few drops drawn off, in particular, had the most sickening fœtor. This condition suggested gangrene of moist tissues. We were unable, even by pressure on the hypogastrium, to empty the bladder. It was thought that the difficulty arose in part, at any rate, from the blocking up of the eye of the instrument by blood-clots, but there was evidently a further cause. On one or two occasions, when the instrument was in the bladder, there appeared to be a sort of clonic spasm, as if the muscular wall contracted forcibly, but with regular and short intermissions. Each contraction expelled a few drops of urine along the catheter. We came to the conclusion owing to the nature of the stuff drawn off that, in reality, the difficulty was due to sloughing of the mucous coat of the bladder, and to anticipate at once the results of the post-mortem investigation I may say that this view turned out to be correct. The whole of the lining membrane of the bladder had sloughed and lay loose in the cavity, forming a complete cast. At the post-mortem the bladder was opened from above, and then, on passing a steel instrument in the ordinary way, we found that the loose slough was lifted up on the point and almost entirely prevented any escape of urine; to draw off any, indeed, it would have been necessary to thrust the point of the instrument

through the slough and into the cavity containing the urine. Even suprapubic puncture might have failed to draw off the water towards the end of his life. The bladder for the last few hours of life was considerably distended, but the man refused to have any operation done to relieve it, and before the distension was at all serious the patient was almost moribund. I feared that suprapubic puncture through the rotten wall of the bladder might lead to extravasation in the pre-vesical cellular tissue, with the inevitable result of sloughing cellulitis. This, however, would not have happened, I think, as the parts, post mortem, were found to be all matted together. The omentum was firmly adherent over the bladder, and a much distended coil of the sigmoid flexure and first portion of the rectum was adherent firmly to the posterior wall of the bladder. The perineal section would have enabled us to extract the whole of the slough with ease, and, no doubt, would have improved the condition, though it could not have materially prolonged life. The case, however, forms a considerable argument for the propriety of making a large opening into the bladder through the perinæum in cases of the kind where there is much vesical trouble. What we took to be clonic spasm of the bladder was in reality due to the valvular action of the slough. Urine no doubt escaped when pressure was made through some rent in the slough, and then the opening was closed again by the loose shreds—the same phenomenon is often seen when a large tealeaf chokes up the opening of the spout of a teapot.

The wall of the bladder was so excessively rotten and friable that it tore as easily as wet blotting-paper. Yet, even in this state, perforation is most unlikely to occur from the natural contraction of the bladder. If the slough had extended right through the walls, the urine would have escaped into the peri-vesical tissues; but, in all probability, no intra-peritoneal rupture would have occurred. The mere weight, however, of a catheter lying on the viscus might have caused a perforation.

With regard to the changes in the alimentary tract, there is little to be said. At first, constipation was obstinate, and when with repeated enemata we got the bowels to act, the motions were very foul, showing evidence of extensive fermentative changes going on in the intestine. The mucous membrane, indeed, throughout the alimentary

canal was undergoing changes similar to, though not of so severe a character as were noticed in the bladder. After a while diarrhoea set in, and the trouble that the nurses had in managing the patient was trebled. Bear in mind that in these cases the diarrhoea is often of the kind called "spurious," and may be associated with an immense fecal accumulation in the bowel. Just as incontinence of urine is the sign of an over-distended, and not of an empty, bladder, so may this variety of diarrhoea be associated with a loaded and not an empty bowel. Enemata and purgatives are required, not astringents, to remedy the condition.

The patient had what may be called an acute bed-sore. He was put on a water-bed at once, and there was no reason to think there was any pressure on the upper parts of the buttocks at all. Yet, almost immediately, the condition of the skin on either side of the sacrum looked threatening, and in a very short time two symmetrical bedsores formed. Such sores may sometimes extend to an amazing depth. The whole of the tissues overlying the bone may slough away, and with extreme rapidity; and down at the bottom, when the sloughs come away you may find the sacrum or ilium already diseased. Indeed, the destruction has often proceeded further than this, and there are cases where the cerebro-spinal fluid has flowed away for a long period of time, until death in fact. The outlook was very bad, and we expected that the trouble would further extend in depth. On the contrary, however, a superficial slough separated away, and a day or two ago the sore already, although it was terribly extensive, began to mend rapidly. Again one of these characteristically rapid changes. The progress in these cases is most uncertain.

I need not dwell on the treatment of either of these cases save with regard to the question of operation. The treatment of the first case—the man who was in the hospital for more than twelve months—was almost vested in the sister of the ward and the nurses who worked under her. If you watched the case you might well have learned what good nursing really means, and what good nursing can achieve. The patient elected at last to go home, and refused to become an inmate of the infirmary. Within a day or two of his leaving the hospital the troubles—bedsores and the like—against which the

nurses had battled successfully for so long set in, and his end cannot now be far off.

In this case operation on the spine had been suggested, but declined by the patient. In the second case operation was performed a few hours after admission.

The arguments in favour of operating were not strong, but yielding to the views of those who urged that the operation itself was not a grave matter, that there was at least a possibility of doing good, that the case was obviously hopeless, and that it was perfectly justifiable to operate, I took the course, and I took full responsibility upon myself for doing it. The operation is generally described as trephining the spine. But I would advise you at the outset never actually to trephine the spine; the trephine is a most unsuitable instrument to use in these cases. The operation which we proposed in the first case and performed in the second, was laminectomy, as it is called. There was no irregularity of the spinous processes, but there was tenderness over the upper dorsal region. It was obvious that the paraplegia was complete, and that the cord was gravely injured. The fear was that this condition was not due to the compression arising from extravasated blood. Pressure on the cord from blood is, I think, more often met with in violent sprains and wrenches than in dislocation or fracture of the spine. I cut down the upper dorsal region where I expected to find the mischief, making an incision five inches in length over the spine, directly in the middle line. Having exposed the spinous processes, I felt one after another of them to see if they showed any signs of preternatural mobility, or if they yielded crepitus. There was no crepitus, but the second dorsal spine was unduly movable. The muscles attached on each side of the spinous processes were dissected off and the laminae exposed. The spinous process of the second dorsal vertebra and the laminae were then cut away with bone-cutting forceps without inflicting any injury on the cord. I drew away a sharp piece of bone the size of the tip of my little finger, which was evidently due to a fracture, but the fragment did not seem to press on the cord. I could not get at the body of the vertebra, nor probably, as you may judge from these specimens, could I have dealt with it satisfactorily without injuring the cord. The operation

then was incomplete, but in reality there was no discoverable condition that could have been relieved by operation. The wound healed well, and no ill effects followed, though no good was done. At the best laminectomy is but a partial operation, even though you succeed in reaching definitely the bone which is pressing on the cord. The mischief to the cord, in the vast majority of cases, has been done, and the destruction is so great that recovery is out of the question. Mr. Cline many years ago said that to operate on these cases was really comparable to trephining the vertex of the skull when you believe that a mass of bone is sticking into the brain at the base of the skull, or, one might add, to trephining the skull when the brain is pulpified by the injury. There is, so to speak, brain tissue to spare, but this is not the case with regard to the spinal cord.

I have described to you the operation as it was performed, and without reference to the precise knowledge of the condition which is obtained by the post-mortem examination. Two points, I must frankly admit, were strongly in evidence at the post-mortem. One was that the operation was not performed at the right level in the spine. We judged by the symptoms, and these pointed distinctly to an injury of the cord at the level at which we operated; but when the spinal column has been violently wrenched, or forcibly bent until some of the bones give way and are fractured, when, in short, we get the condition which is spoken of as "dislocation with recoil," it by no means follows that the injury to the cord will correspond with the fracture of the bone. In this case the injury was much higher up in the cord. It is true that corresponding to the level at which the vertebra was broken, the cord was extensively damaged, being in fact reduced almost to a pulp; but it is obvious that so violent a stretch of the cord will produce lesion enough to cause paralytic symptoms at a considerable distance above or below—but most likely above—the lesion of the spine. Then, in the absence of any irregularity of the bone, the symptoms will be most misleading. It was perfectly evident at the post-mortem that I could not really have done any good by local treatment of a surgical nature to any part of the damaged cord. There was no displacement whatever to be met with along the spinal column, and

at the post-mortem it was only after careful dissection of the parts, and close investigation, that the line of fracture was discovered. The hope that in such a case symptoms are due to pressure of blood-clot on the cord is a very slender one, and these patients are really likely to do quite as well without any operation. The conditions are quite different, from the surgical point of view, in the case of the brain and of the spinal cord. The term "trephining" the spine has, I am inclined to think, misled people into supposing that the two conditions have more in common than is actually the case. Very seldom indeed will you meet with a case where a portion of the lamina has been driven right into the spinal cord. In such a case it would, no doubt, be right to remove or raise up the offending piece of bone; but even then, although the mechanical condition be completely relieved, the benefit will probably be far less than in a case of a compressed or wounded brain. Myelitis may readily supervene, and in any event the recovery of the cord to a sufficient degree to discharge usefully its functions would be highly improbable. In an accident or injury where a severe blow had been received on the back from a sharp-edged instrument, it might be proper to cut down on the part in the hope of doing some good. But in a diffused injury, such as results from fall from a height, it is almost certain that the fracture will be found in the bodies of the vertebræ, and that if the lamina is broken at all the fragments will not be found to compress the cord. You will find many specimens in our museum and elsewhere in which there is fracture and dislocation of the body of the vertebra. Here, for example, is one (specimen shown) in which a portion of the body of the vertebra projects into the spinal canal. In such a case, if you could have removed the offending bone and prevented it from irritating the cord, you would have done good. But the bone is in a situation where you cannot get at it.

There were not a few surgeons who hoped at the beginning of the Transvaal War that a useful field might be found for spinal surgery. Such hopes, I venture to think, have been altogether disappointed. Surgery under the conditions of war could do even less for these unfortunate sufferers than it could in severe penetrating abdominal wounds, and of the latter, at least a fair pro-

portion recover when they are left altogether alone.

At first sight one might think that in these cases there really would be a chance of success. I saw many cases in South Africa of gunshot wounds of the spine. They were frequent because the wounds were so often received when the men were lying down facing the enemy. I saw one case out in South Africa which was very instructive to me. It was that of a man who was shot through the spine in the upper dorsal region. We could judge fairly accurately the track which the bullet had taken, because we knew the position of the man when he was struck, and we saw the aperture of entrance and exit. It was a clean Mauser bullet wound, leaving little "bug-bite" scars, sure evidence that the bullet had not broken up at all. The course of the bullet indicated that the spinous process and the laminæ might have been struck, but it seemed likely that the bullet had not actually touched the cord, and, it was thought, that possibly fragments of the bone pressing on the cord might cause the paraplegia and incontinence of urine and fæces from which the man suffered. There was a terrible hyperæsthetic area in this case, a symptom that is very marked in gunshot wounds. No operation was, however, performed, and soon after the man died. We had a post-mortem, and followed the track of the bullet from one side to the other. The diagnosis was perfectly right; the cord had not been touched at all; the theca seemed to be uninjured, and the bullet had passed obliquely across the back of the cord and had broken up the laminæ on both sides into several pieces. What then was the explanation of the extensive paralysis? The theca of the cord looked as natural as in health. For the moment, as we looked at the parts we felt that here at last was a case in which operation might have done good. We were soon undeceived. When we came to lay open the theca and exposed the cord we found that over an extent of some three inches the cord was reduced to a grumous fluid mass, utterly and entirely destroyed. How that comes about it is perhaps a little difficult to explain, but the immensely rapid passage of the bullet through the tissues must produce tremendous vibration effects. The cord was not directly injured by the bullet, which had merely passed close to it. Many instances were seen where a bullet passed close to a nerve without

touching it, but yet produced a profound lesion of that nerve, and in the case of the more delicate and friable cord a similar wound was able to disintegrate it absolutely and entirely. If we had taken away every scrap of bone that might have compressed the cord in that soldier the operation would have done no good.

New growths of the cord have been successfully dealt with, and the treatment of these rare cases forms really a brilliant chapter in the history of modern surgery. But in these the symptoms are localised and truly indicate the morbid condition. The huge majority of traumatic cases seem to me to be as far removed as ever from the domain of practical operative surgery.

Gastric Splashing and Atony.—B. Stiller ('Berliner klinische Woch.,' September, 1901) vigorously opposes the dicta of Elsner, who recently deprecated the value of gastric succussion as an indication of atony. According to Stiller's views, there may be atony without muscular insufficiency, and this may be a forerunner of gastropstosis, which condition Elsner admits greatly favours the production of the splashing sound. Stiller therefore argues that the presence of this sign means both ptosis and atony, and says that this triad forms the most constant symptom of nervous dyspepsia. Atony of the stomach means a relaxation of its musculature, which leads to impaired contractility, dilatation, and depression of the lower limits of the organ, with ultimate ptosis. In pure atony only the tonic contraction, and not peristalsis, is defective, but this also becomes gradually impaired so that complete motor insufficiency and gastrectasia may, but does not necessarily, occur. Only rarely is atony a purely local condition dependent on regional causes. In most cases it is due to atony of the entire organism, which leads to enteroptosis, nervous dyspepsia, and general neurasthenia. Such a condition may be called *asthenia universalis congenita*, and of this the atony of the stomach is the most constant sign. The presence of splashing is of the utmost importance as indicating the presence of such a nexus, and its value as a diagnostic aid cannot therefore be underestimated.—*Medical Record*, October 26th.

A DEMONSTRATION ON INFANTILE PARALYSIS.

At the Hospital for Sick Children, Great Ormond
Street, W.C., July 25th, 1901.

By F. E. BATTEN, M.D.

THE subject which I have chosen for my demonstration to-day is that of infantile paralysis, and I propose to show you first a typical case of the disease, and then one or two cases of palsy in children in which the diagnosis is not so clear. Finally, I should like to say a word or two about the pathology of acute anterior poliomyelitis. The first case is that of infantile paralysis affecting the arms. This child was brought to me in November of last year with a history that two months before he had a bad cold for two days, with a rash which was said to be chicken-pox. The mother then noticed that he could not use his arms. He was unable to walk for about fourteen days after the acute illness, and could not hold up his head. A fortnight after the child was first taken ill he began to recover power in his legs, but his arms remained completely paralysed, and little or no improvement had taken place when I saw him in November. One can study the disease very well in this child. You see there is a considerable wasting in the deltoid, biceps, and triceps, and the arm hangs absolutely flaccid. He has still some power in his pectoral muscle. Both arms are almost equally affected. On testing him one finds that the sensation is perfectly normal. We have here a disease coming on acutely, giving rise to a paralysis which is very extensive at first, affecting the muscles of the neck and legs, and passing off and remaining localised after the first month to the arms, and more especially the upper arms. You will notice that he had very fair strength with the small muscles of his hand, and also with the flexors and extensors of the wrist. It is exceptional to get the small muscles of the hand paralysed unless the whole arm is involved.

There are some interesting points with regard to the distribution of the palsy in infantile paralysis. Knowing as one does that the disease is of spinal origin and affects the anterior nerve-cells, it might have been supposed that certain groups of muscles corresponding to the segment in which the disease had occurred would be affected. For instance,

as you know, in the fifth cervical segment the following muscles are represented—the supra- and the infra-spinatus, the rhomboids, the deltoid biceps, and supinator longus; but it is a curious fact that one seldom gets the accurate localisation of the lesion that might be expected. It is quite common to find that the disease will pick out certain muscles which do not correspond to the segments involved. For instance, there is sometimes affection of the infra- and supra-spinatus, the triceps and extensors of the wrist. The infra spinatus is represented in the fifth, the triceps represented in the sixth, and the extensors of the wrist in the seventh cervical segment. The explanation of this I hope to be able to show you when dealing with the pathology.

With regard to this boy, he had at the onset loss of power in his legs. There is at the present time neither atrophy nor weakness in his legs. The question which arises is, was the weakness in his legs due to an affection of the grey matter of the cord in the lumbar region, or was it due to the lesion taking place in the cervical region, and in that way affecting the cord so seriously at that level, that it interrupted the path for the legs in the pyramidal tracts? Had that been the case, it might have been expected that subsequently there would be some degeneration in these tracts, and that would have manifested itself in a more or less rigid condition of the legs, increased knee-jerks, ankle-clonus, and an extensor plantar response.

The weakness of the legs passed off rapidly, and therefore, if the weakness were due to this cause, it was merely a temporary loss of function from which the pyramidal fibres rapidly recovered, and left no permanent injury. It is, however, much more probable that the loss of power in the legs was due to a temporary vascular disturbance in the grey matter of the lumbar region. But it is worth examining this case in order to see if there is any evidence of affection in the pyramidal system. His legs are well nourished and fat, and the knee-jerks are retained, and the plantar reflex gives a very definite flexor response. Thus there is no evidence of any affection of the pyramid, and probably in the first instance the disease was one of the grey matter, a general vascular disturbance which had its focus of greatest virulence at the level of the fifth cervical segment. It occasionally happens that the onset of the disease is ushered in with con-

vulsions, but this is by no means common. The muscles most commonly affected are those of the legs, and the anterior tibial group are those most liable to suffer.

When, however, the arm and leg are affected on the same side, it becomes necessary to distinguish the condition from one of hemiplegia; and the flaccidity of the limb, the wasting of the muscles, the absence of knee-jerks, and the altered electrical reaction in infantile paralysis serve to distinguish the condition from one of hemiplegia in which the limbs are rigid, the muscles but little wasted, the knee-jerk exaggerated, and the electrical reaction unaltered.

The next case is one of considerable interest, especially from a diagnostic point of view. The child, *æt.* 2½, was brought to me in November last year with a history of weakness in the right leg, which was first noticed when he should have begun to walk. The child has had weakness of the left arm, which was noticed immediately after birth. The birth was natural, but very rapid, the labour lasting only twenty minutes. The child fell from the bed on to the floor. The mother had no illness while carrying the child. When I first saw the child it was rather poor and thin; it could talk, but could not walk. The right arm was well developed, but the upper portion of the left arm was wasted and paralysed. You will notice the boy has weakness of the deltoid, biceps, and also of the supinator longus of the left arm; it is probable, therefore, that the child has had some injury to the fifth cervical root or segment. The condition was noticed immediately after birth, and therefore must have been due either to a *præ-natal* condition or to injury at the time of birth. The right arm is moved normally. Nothing was noticed to be amiss with the leg at the time of birth, and no weakness was observed until the child began to try to walk, and it was then the mother found that the right leg was extremely limp. The movements of the left leg are quite normal. There is very marked wasting of the muscles of the right thigh. Electrically the anterior tibial and gastrocnemius muscles of the right leg do not react to faradism, neither do the deltoid, the biceps, nor the supinator longus of the left arm. The affected limbs are cold and blue, resembling the condition of limb seen in anterior poliomyelitis. With regard to the knee-jerks the left is

present, but that on the right side is very difficult to obtain. The plantar reflex is of the flexor type. To sum up the points in this case, one has an extremely weak and flabby left arm, dating from the time of birth. A similar weak and flabby condition of the right leg, dating from the time when the child should have walked. The limbs are blue and cold, and there is loss of electrical reaction. There is no alteration in sensation. The diagnosis, however, is by no means clear in this case, the appearance of the limbs, the character of the reflexes, and the electrical reaction suggest that one is dealing with an anterior poliomyelitis. It seems almost impossible to decide whether this was a *præ-natal* condition, or whether it was due to the fall which the child had at the time of birth. All that one is certain of is that the child has affection of the cells of the grey matter of the anterior horn, not only in the cervical region, but also in the lumbar region.

The next case is that of a child two years and five months old. I first saw the patient ten months ago, when it was brought for sudden loss of power in the left arm, which had occurred four days previously. The wrist was dropped, but the child could walk fairly well. You will notice now that as the child walks she drags the left leg, and she holds the left arm to the side. When I first saw the child there was no weakness of the leg, but very marked wrist-drop, and the question arose whether that was due to an infantile paralysis, or whether there was some other lesion; and at first I was rather inclined to imagine that there had been some injury to the musculo-spiral nerve. On seeing the child a few days later, however, it was obvious that he was suffering from hemiplegia, which had affected his arm far more than his leg. The wrist-jerk on the left side was brisker than on the right. The same is true with regard to the triceps jerk. The upper arm he moved fairly well. He had no ankle-clonus. The last time I examined his plantar reflex he had a very definite extensor response on the left side, but at the present time it is flexor. He now has some in-co-ordination of his left hand, and the tendency to athetotic movement. That is a movement which is never seen in infantile paralysis. With regard to the variability of the plantar reflex in this child, one may say that variations in this respect are most common in young children, but the alteration from extensor to flexor may indicate that recovery

is taking place in the affected leg. The case is one of sudden hemiplegia in a child. It is always difficult to state definitely what is the cause of hemiplegia. The suddenness of the onset would suggest some vascular lesion, and that vascular lesion is most probably thrombosis, affecting a small area limited to the fibres passing to the arm. I have examined one or two such cases in which the focus has been accurately limited to a very small area, situated sometimes in the pons, sometimes in the internal capsule, apparently just picking out the fibres of the pyramid which are passing to one part of the body.

This next case is that of a child who was brought to me some little while ago because his back was growing out. It is eleven months old, and the mother said she did not notice any prominence on the back until it was seven months old, but the left hand had been paralysed since birth. The left hand is in a flexed position, and the child is apparently unable to open it. The arm can be moved, and the movements are performed better on the right side than on the left. This child is extremely small for eleven months old, and one finds, on looking at its back, that there is a marked prominence on the left side. That is formed by the displacement of the ribs, and if one places one's hand on the spine of the vertebræ one finds a marked deviation with a concavity of the right. This concavity is most marked in the upper dorsal and lower cervical regions. In the lower dorsal region the spines are in their normal situation. Now, if you will look at the child, you will see that the palpebral fissure on the right side is considerably larger than on the left, and also that the right pupil is larger than the pupil on the left side. And you will notice also that there is a marked alteration in the character of the skin on the right side of the face compared with that on the left. This is apparently due to the child sweating on the right side more than the left, and it forms a very marked line down the middle line of the face, and the child has been often observed to sweat on the right side and not on the left. With regard to the legs, it is difficult to make the child move them. One may stimulate them, but it cannot be made to do it in any other way. If one examines the reflexes, one finds that the knee-jerk on the right side is brisker than the reflex on the left, but there is no clonus. With regard to the plantar reflex,

there is a double extensor response, which in a young child of this age has but little significance. With regard to the arms, there is a brisk arm-jerk on the right side, not on the left. The right arm has a tendency to be more rigid than on the opposite side. There is one other phenomenon which I want to demonstrate here if I can, and that is one in which the diagnosis of this case is of great interest. When the left leg is pricked the child shows no evidence of feeling pain, while, when pricked on the right side, the child immediately cries. What is the significance of these various phenomena which we have noticed? There is greater loss of power on the right side of the body than the left, there is apparently loss of sensation to pain on the left side of the body, there is a wider palpebral fissure on the right side, a dilated pupil, and sweating on the same side of the face, and there is a curve in the spine with concavity to the right.

With regard to the arm, there is a certain amount of rigidity in the right arm, and there is evidently greater weakness in the right arm and right leg than the left. It is a well-recognised fact that a spinal lesion affecting the centre of the cord, and especially in hæmorrhage into the grey matter of the spinal cord, gives rise to what is known as a Brown-Sequard paralysis—*i.e.*, there is loss of power on one side of the body and loss of sensation on the opposite side, the loss of sensation being generally limited to heat, cold, and pain, while tactile sensation remains normal. How does that fit in with the other symptoms which are present? There is the dilated pupil on the right side, retraction of the eyelids from contraction of the fibres of Muller, and sweating on the right side of the face. These are manifestations of irritation of the sympathetic. The sympathetic fibres pass down the cord and emerge about the level of the first dorsal root, and then pass up the cervical sympathetic to the face and eye. By irritating the sympathetic retraction of the fibres of Muller and dilatation of pupil are produced, but not sweating. It is, however, not uncommon to see unilateral sweating in connection with affection of the sympathetic. In this child there is evidence of some spinal lesion at the lower cervical and upper dorsal region of the right side, and it seems not improbable that that lesion is an hæmorrhage into the spinal cord, and that hæmorrhage was caused by the difficulty at birth.

Now one or two words with regard to the pathology of infantile paralysis. For the sake of convenience the disease may be divided into three stages. The first stage is that of acute inflammation, the second stage that of destruction and removal of the resulting products, and the third stage that of contraction. The view which was originally taken was that it was a direct cell infection, a direct action of toxin on the cell. Most observers at present agree that there is a vascular lesion. The question arises, is it a primary vascular lesion, or is it secondary to some infective organism or some toxin which gives rise to thrombosis? I would ask you for a moment to pay attention to the vascular supply of the cord (drawing a transverse section through the spinal cord). The spinal cord is supplied by the anterior median vessel, being derived from the two vertebrals, and this vessel passes down the anterior fissure of the cord. The vessel then passes into the anterior fissure and divides into two branches, and these again divide and supply the anterior fissure of the cord, such as I depict here. Further, along each root there comes in a vessel; these vessels anastomose with the anterior median vessel, so that this vessel, instead of getting smaller as it passes down the cord, remains of the same size. You will notice that the anterior horn is practically only supplied from the anterior median vessel. It is important to remember that, because the disease tends to limit itself to the one part of the cord. The first drawing I show you is of the spinal cord taken from a child who died fourteen days after the onset of the disease. Round each of the vessels there is very extensive peri-vascular injection, and in certain parts there are extravasations of blood. There is extreme congestion of the spinal cord in the area of the grey matter.

Passing, then, from the early condition, one comes to the case of a child who died three months after the onset of the disease. Here is a transverse section of the spinal cord stained by Marchi's method, showing destruction of the cord in the region of the anterior horn. It will be seen that the whole of the anterior horn is picked out, leaving the posterior horn and the white matter normal. I would ask you to compare the area of softening in this specimen with the area which is supplied by the anterior median vessel. You will see that that almost exactly corresponds with the

area supplied by that vessel. The question is: Is that an inflammatory process, or is it due to a thrombosis of that vessel? Everybody will agree that the vessels are thrombosed, but many observers look upon this as a condition secondary to an inflammatory process. If one was shown such an area of softening in the brain one would at once ascribe it to thrombosis of a small vessel. If it is an inflammatory condition, why should the disease limit itself almost exactly to the area supplied by one vessel? Other forms of inflammation do not limit themselves to the anterior horn, but extend into the posterior horn, and also into the white matter, and infantile paralysis is one of the few diseases which limits itself exactly to the anterior horn. Other observers regard the poison as having a special selective action on the anterior horn, and just as lead picks out the extensor muscles of the wrist, so the specific poison of infantile paralysis picks out the anterior horns. The view that the disease is caused by thrombosis of certain vessels is that which seems to me to be most supported by pathological evidence, but it has yet to be shown whether such thrombosis is dependent on some one specific infection or is due to vascular changes which may occur after various kinds of infection.

Now we pass to a still later case, namely, to the condition as found two years after the onset of the disease. This section was taken from the lumbar region. You will notice that the anterior horn on the one side is considerably smaller than the other, and also that the anterior columns are considerably paler than the posterior. A considerable amount of degeneration takes place in the antero-lateral columns in the fibres which are ascending from the grey matter of the anterior horns. Here is a representation of the sciatic nerve in section, and the fibres which are shown here are large ones. The mixed nerves are made up of a large number of sensory and motor fibres, and in infantile paralysis the motor fibres atrophy and the sensory fibres remain normal, so that the normal fibres which you find in any given nerve are the sensory ones. In the muscle you will notice that some fibres have undergone atrophy, but a few good-sized fibres remain. One other point in connection with the muscles is of interest, and that is the bodies known as muscle spindles remain perfectly normal, and the explanation of this is simple, for they have been shown to be from fibres derived from the posterior roots.

A DEMONSTRATION OF CASES AT CHARING CROSS HOSPITAL.

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F.R.C.S.

Senior Assistant Surgeon to Charing Cross Hospital and
to the Victoria Hospital for Sick Children.

(Concluded from p. 48.)

OCCIPITO-ATLANTAL MENINGOCELE.

The next case is a very good example of occipito-atlantal meningocele. Pure meningoceles are distinctly uncommon. The occipital is the commonest region for meningocele, and certainly the commonest for large meningoceles. We are too apt to call all these swellings meningoceles. You may find one of three conditions. The first is, where there is protrusion of the membranes of the brain, filled with serous fluid. This case is a very good example of this type. The commonest condition is the second variety, meningo-encephalocele, where there is a protrusion of brain substance in addition to the cerebro-spinal fluid. The third variety is hydrencephalocele, where a part of the brain substance is included in the sac, and this part of the brain substance is distended by a central cavity, originally, or still, in connection with the lateral ventricles. Of course it makes an immense difference with regard to treatment which condition you have to deal with. If you have a pure meningocele, such as the present one, and you have no doubt whatever that that is what it is, for you can press it back with your finger, and can feel the gap between the occipital bone and the atlas, you will find such cases comparatively simple to deal with. Under antiseptic precautions one taps the meningocele and ligatures the neck of the sac, and I always, when possible, close the foramen in the bone, usually by transplantation of a little piece of bone, or by turning down a flap of bone from the skull. Where you have a considerable piece of brain substance, whether it be meningo-encephalocele or whether it be hydrencephalocele, whether the brain substance is solid or whether it contains a cavity, the condition is very different, and any interference with the protrusion is a direct interference with the brain substance itself. Therefore the majority of such cases are certainly best left alone. In many cases the meningocele is a huge swelling, sometimes it is larger than the cranial

cavity itself. When you get such a case as that it is, of course, hopeless to try to push back the structures, as you would increase the intra-cranial pressure so that the child would die. It is only moderate sized meningoceles that are amenable to surgical treatment. You expect a meningocele to pulsate, but frequently a meningocele does not pulsate. I have now reduced the meningocele entirely. If you can reduce a meningocele it means that it is very amenable to surgical treatment. I intend to operate on this child when it gets a little older.

GUMMATA.

I have next to show you two very good examples of gummata. They are both cases in which the diagnosis of gumma has been confirmed by the result of treatment. One is a young fellow who was brought to me only last Tuesday with a large horridly fetid ulcer in the cheek, and on examination there was found an extreme amount of induration. Dr. Stansfield, who brought him to me, said the patient when he first consulted him complained of a swelling in the cheek, that a tooth was evidently cutting into it, that this tooth had been removed, but still the ulcer continued to grow and discharge. On looking at it the first thing I noticed was a wash-leathery slough, distinct and typical. Further, the man being only thirty-four, we did not think it was malignant. On closely questioning the patient I found that, though he had bitten the part several times, and though the tooth had been causing irritation, there was no doubt that the swelling came before the tooth injured the part. It was not, therefore, a case of dental ulcer caused by displacement of a tooth or by a sharp piece of decayed tooth excoriating the cheek, but it was the case of a swelling which got into the way of the tooth owing to its projection inwards. I immediately put him on twenty grains of iodide of potassium and a drachm of liquor hydrarg. perchloridi, three times a day, and I can see how very much better he is to-day. This case illustrates the fact that gummata may occur practically anywhere; I do not think there is a single region of the body in which I have not seen a gumma. You can feel there is still a good deal of induration on the outer side. The induration in this case was partly due to the gumma and was partly of septic origin. You can see the ulcer from

inside the mouth. The cheek is not a common situation for gumma. The features which led me to the diagnosis were, first, the distinct amount of washleather slough, and, secondly, the history that the swelling had been bitten by the tooth because it got in the way of the tooth, and, thirdly, the confession of syphilitic infection five years previously.

Here is a gumma in another unusual position. It is a subcutaneous gumma above and external to the external angular process of the frontal bone. It was very deceptive. When the patient was sent to me it was with the question whether it was a malignant pustule, the reason being that it was covered by a circular black slough. At first I had some considerable doubt about it. On removing a small piece of the slough we came down upon the washleather slough. It has almost entirely peeled away, and the whole ulcer has all but healed. The woman has had twenty grains of iodide of potassium three times a day. In cases like this I continue the iodides for at least a week after all symptoms have disappeared, and then give the patients iodide occasionally as a precautionary measure. As a matter of fact, one finds those patients who suffer from gummata of the skin or subcutaneous tissue generally escape gummata in the viscera and central nervous system.

CHRONIC ARTHRITIS DUE TO TRAUMATISM.

The next case I want to show you is one illustrating a chronic arthritis due to traumatism. One is far too apt—at any rate I am—to conclude that every case one sees of chronic painless swelling of the knee-joint is tubercular. When this patient first came under observation we felt almost certain that he had a chronic tubercular knee. We should have been certain if it had not been for one fact, namely, that it followed a somewhat severe injury. The patient is a foreigner and an hotel waiter, aged about forty five years. Eighteen months ago he received an injury to his knee, which was said to be dislocated. He was carefully treated for this, but after some weeks the knee began to swell, and it slowly and steadily got worse. Twelve months after the accident the patient came under my care. I had seen him two months before that, just prior to starting on my summer holiday. I found there was a certain amount of fluid in the joint, and the whole synovial membrane was very much thickened.

There was considerable limitation of movement, but not so much as you would expect in a tubercular knee. I thought it was a case of chronic traumatic synovitis and ordered a back splint and complete rest in bed. When I saw him in September there was no doubt suppuration had occurred into the knee-joint. The question was still rather doubtful whether it was a tubercular knee or not. The point which made me think it was probably not tubercular was the amount of movement which he had was very much more than one would expect in a tubercular knee. Therefore I determined, before doing anything further, to make an incision on each side of his knee and let out the pus, and then wash out the joint. This I did, and you can see where the incisions were made. A lot of broken-down debris and puriform stuff was washed out, and drainage-tubes inserted. This was about September 18th, and he was kept in hospital three weeks. The pus removed was examined for tubercle bacilli, but none were found. Further, Dr. Eyre inoculated guinea-pigs—which is the real test for tubercle—but none of them developed tuberculosis. So we may take it for certain that the condition is not tubercular. I want you to notice how good the result has been. You will observe how well he walks. There is one thing I want to call your attention to, and that is that the joint feels weak and “wobbly.” That is a thing one notices very markedly when effusion into a joint has lasted for months, for the capsule and ligaments get stretched, and even though you remove the effusion the joint is practically never itself again—the ligaments remain stretched and the capsule remains distended. That is a very good reason for not allowing an effusion into a joint to remain too long. If an effusion has become chronic it is surely very much better to aspirate and remove it instead of allowing the capsule to be distended and the ligaments to be markedly stretched.

LIGATURING POSTERIOR TIBIAL.

There are two very interesting cases in the Robertson Ward which I wish to show you. The first is a somewhat unusual one. The patient was admitted here on January 25th, and I ask you to particularly notice the date. He had been run over, but does not know by what. He is himself a driver of a van. The vehicle passed over his leg.

He had a good deal of hæmorrhage and much ecchymosis, but there was no fracture. The house-surgeon who admitted him states that he was in a state of considerable collapse. For the first week he was in the ward nothing of importance happened, except that the skin on the outer aspect of the leg looked very sloughy, and it actually did slough in certain places. On February 1st he suddenly had pain, and Mr. Bonney, the house-surgeon, looked at his leg and found it was becoming very tense and distended. Accordingly he telephoned to me. It seemed to me rather unusual that arterial hæmorrhage should occur about a week after the injury, and I replied to Mr. Bonney that I was not anxious to come down and cut into a huge infiltrated calf like that, because one was very likely not to be able to find the bleeding vessel. I, therefore, asked him to put on elastic pressure. Pressure was put on, and two hours afterwards Mr. Bonney reported that the limb was distended almost to bursting. It was then clear that some vessel had given way; one could just feel pulsation in the anterior tibial, but not in the posterior tibial, but the whole swelling pulsated. I then saw that there was nothing to do but to cut down, hunt for the posterior tibial, and tie it. I made an incision along the inner aspect of the calf, from just below the head of the tibia, and turned out one and a half pounds of blood, clotted and liquid. I exposed the posterior tibial artery, and I felt it was pulsating; I felt again a little lower down and found it was pulsating, and I, therefore, followed it down until I came upon a spot where pulsation suddenly stopped. That was the point from which the blood was oozing up. This was only about a couple of inches above the internal malleolus. Without very much difficulty I found the bleeding point in the posterior tibial, doubly ligated the vessel, and divided it across. The patient has done very well since the operation, and his temperature has fallen practically to the normal. We have had no recurrence of bleeding, and things look very promising. You will notice, in the first place, what an enormous wound I had to make from the internal tuberosity of the tibia to the malleolus. But there was nothing to indicate where the bleeding point was, except that we could not feel the pulsation in the posterior tibial just above the inner ankle. The blood had become effused under the deep fascia and had tracked up-

wards. There was little detachment of muscle at the operation. The only muscular fibres I cut through were those of the origin of the soleus from the inner margin of the tibia. This I stitched back into position, and I think the limb will probably be almost as good as ever it was. I sewed up the wound tightly, and then put it in a drainage-tube. I can scarcely describe to you what the limb looked like before the operation was done.

GENERAL SEPTIC PERITONITIS.

The next case is a very interesting one indeed. Last time I was able to show you two cases of general septic peritonitis in the Golding Ward that were both apparently recovering. One of those patients, in which the peritonitis was due to the bursting of a gonorrhœal pyosalpinx, is now well. The other patient, unfortunately, died three months later of intestinal obstruction, due to adhesions caused by the general peritonitis. We opened her abdomen and washed it out thoroughly, but the intestines were so involved by the gluing of fibrinous adhesions that she suffered from more or less chronic intestinal obstruction until she died. This present case teaches us many a lesson. He was admitted here on Monday afternoon. His history is as follows: At about twelve o'clock on Monday he was seized with a bad pain in the belly. The pain continued to be so severe that he came to the hospital at two o'clock, in a cab. He had only had his pain two hours. The house surgeon saw him and found his pulse and temperature were both absolutely normal. Now, in three cases out of four a man coming and saying he had pain in his belly, with an absolutely normal pulse and temperature, would have been given some medicine and would not have been kept in. But it seemed a genuine case and the house surgeon wisely admitted him. When he came in, as you see from the chart, his temperature was 99°. Two or three hours later it was 100°; next time it was 101°, and at twelve o'clock at night it was 102°. The pulse, which at first was normal, had become 144 by midnight, when I was sent for to see him. His abdomen was distended, he had vomiting, the vomit being greenish stuff, which later became a blackish brown. The abdomen was rigid and scarcely moved at all, his tongue was beginning to get dry, and he had the appearance of a man in an absolutely poisoned state. The pulse was gain-

ing in rapidity at the rate of something like ten beats every hour. He had passed no flatus, and he was getting into a bad state. There was scarcely any doubt that he was suffering from general septic peritonitis, and it was in all probability a perforative case. There was no loss of liver dulness, but that is a very deceptive sign. I have found liver dulness normal with a good sized perforation; and, on the other hand, I have found it absolutely lost at the front and side owing to tympanites, due I think, to intestine getting between the liver and the abdominal wall. So the presence or absence of liver dulness is a thing which must not be taken as an absolute guide. I think we had no doubt that there was here general septic peritonitis, but where it started from there was nothing to indicate, except that there was some extra tenderness over the appendix region. I cut down upon his abdomen, expecting to find a commencing general peritonitis. To my astonishment, as we cut through the abdominal wall, all the layers of the abdominal wall were infiltrated, and clearly there was purulent inflammation, which must have been going on for some time. When we opened the abdomen a large quantity of pus escaped, probably almost a pint. I passed my finger in different directions, got it right down into the pelvis and up to the liver. I passed it inwards and found there was a faint barrier, but it was only a very partial one. It therefore became certain that he had had quiescent appendicitis that had given him no trouble at all. That pus was obviously of more than twelve hours' duration. Having made our incision and evacuated the pus, I searched for his appendix and removed it, as it was perforated and contained four or five soft concretions, and its mucous membrane was ulcerated. We then proceeded to wash the abdomen out until the fluid returned clear. One tube was put at the bottom of his pelvis, and it is still there. A counter opening was made in the right loin and a tube inserted there to drain the kidney pouch. Another tube was brought out at the abdominal wound. As it is only four days since the operation was performed one cannot say definitely how the case will go on. I would like to call your attention to the fact that his temperature was 102° at the operation, next night it was 102° , the next day it fell to 100.6° and the next day 100° . But in my experience the temperature in septic peritonitis is no guide whatever. The worst cases of general peritonitis I have seen have been those where the

temperature was subnormal. I dread to get a temperature of 96° and 97° in general peritonitis far more than I dread one of 102° or 103° . 102° indicates a healthy reaction of the organism to the microbic infection, whereas a depressed subnormal temperature generally means that the patient is thoroughly poisoned by the septic infection. What is your real guide? It is the pulse. This, especially after operation, scarcely ever misleads the surgeon. I feel hopeful that this man is going to do well, in spite of the fact that he had general septic peritonitis, and notwithstanding that at one time we would not have given him a chance in 100. His temperature is now lower, the vomiting has stopped, he has passed flatus, and the pulse has fallen from 158 to 120. You will notice that the fall in the pulse rate is a steady one—158, 144, 140, 132, 124, 120, and it is now 112. This case illustrates the advantage of washing out the peritoneal cavity until the fluid returns clear, after you have removed the source of infection and given the freest drainage. A few years ago outlets for two additional drainage-tubes would have been deemed unnecessary, and yet it is free drainage which gives these patients their only chance of life.

But the chief lesson that this case teaches me is that this man thought himself well twelve hours before his abdomen was opened and found to contain a large amount of pus. Also, for certainly two hours after perforation into his peritoneal cavity had occurred the man's temperature and pulse were normal. It emphasizes more than ever that these abdominal cases must not be left. If a man has urgent abdominal symptoms or anything pointing to perforation, he needs to be seen every two or three hours at least, because a very sudden change for the worse may come over him. At twelve o'clock in the day this man was in ordinary health, and then suddenly he felt an agonising pain in the abdomen, caused by the bursting of the appendix abscess into the peritoneum. This man will owe his life—if he does recover—to early operation. Nothing is more instructive than the difference in prognosis when you have an early operation compared with a late one after perforation. I have operated on two cases of perforated gastric ulcer within twelve hours after perforation. They both recovered. I have operated on four at more than twenty-four hours after perforation, and they all four died. If you wash out the noxious material before it has time to thoroughly infect the peritoneal cavity your result is good, but if the peritoneal cavity has been thoroughly infected the patient's chance of recovery is exceedingly small. A thing which was probably very much in his favour was that the intestine only happened to be a little distended. Abdominal section for peritonitis with distended and therefore paralysed intestine is practically hopeless.

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CHAPTERS FROM THE TEACHING OF DR. FREDERICK ROBERTS.

ON SKILLED EXAMINATION OF ABNORMAL ABDOMINAL CONDITIONS.

IN a previous communication to "THE CLINICAL JOURNAL" I attempted to give a comprehensive sketch of the methods of skilled examination applicable to the chest and abdomen, and of the objects for which they are severally employed. As regards the abdominal region, in addition to its general examination from without by the ordinary physical methods, certain of its contents have to be investigated in particular ways, on the lines then indicated; while other more special methods are also not uncommonly called for, such as administration of an anæsthetic, skiagraphy, or operative procedures. I do not intend to discuss the subject further from this point of view, but in the present article propose to deal on general lines with the chief abnormal conditions which are thus investigated. They may be primarily grouped under the following heads:

- I. Conditions of the abdominal walls.
- II. Abdominal respiratory movements.
- III. Intra-abdominal movements.
- IV. General enlargements.
- V. Depression or retraction.
- VI. Local morbid conditions.
- VII. Combined conditions.

I now proceed to consider each of these groups in the order above given.

I. CONDITIONS OF THE ABDOMINAL WALLS.

The abdominal walls should always receive due attention as regards objective abnormal conditions, as they may not only be important in themselves, but give valuable information concerning internal affections. They are mainly determined by inspection and palpation, but other methods are helpful in some instances. In passing, it may be noted that eruptions on the abdomen may be of

significance, such as that of typhoid fever, as well as pigmentation. The more definite conditions to be studied include :

1. Enlarged superficial veins, which may need special investigation as regards their site and distribution, degree of dilatation, and direction of the current of blood. The term "*caput medusæ*" has been applied to a number of dilated veins radiating from the umbilicus.

2. Signs of stretching of the skin, past or present, especially *lineæ albicantes*.

3. Accumulation of subcutaneous fat in various degrees, not uncommonly attaining a great thickness, and interfering with deeper examination.

4. Subcutaneous oedema, especially about the flanks.

5. Subcutaneous emphysema.

6. Chronic thickening of the skin and subcutaneous tissue, often with lymphatic dilatation, giving rise to brawny induration.

7. Relaxed or flabby and toneless abdominal walls in various degrees, often accompanied with more or less wasting of tissues, particularly of the muscles. This condition is not uncommonly due to previous distension.

8. More or less general tension of the walls, indicating some distending pressure from within, especially due to gas or fluid.

9. Undue muscular contraction, either general or local, often brought out during palpation, and leading to a state of tonic rigidity or sometimes painful cramp.

10. Changes involving the umbilicus, such as stretching or obliteration ; eversion or pouching, which may attain a considerable size ; abnormal depression ; thickening and induration ; or special lesions.

11. Adhesion of the walls to internal structures, general or local, through the medium of the peritoneum.

12. Local morbid conditions of different kinds, either originating in the abdominal walls themselves, or resulting from internal affections, in the way of abscesses, sinuses, ulcerations, growths, etc. It is always important to note the presence of any enlarged lymphatic glands.

13. A split or deficiency in the aponeurotic or muscular portions of the walls, varying in seat, dimensions, and form, and allowing herniæ to protrude under the skin.

II. ABDOMINAL RESPIRATORY MOVEMENTS.

The abdominal respiratory movements should always be carefully noted when examining this region, and they may afford signs important in themselves. Thus they may be entirely absent or abnormal ; or during the act of breathing a friction-fremitus or friction-sound is sometimes brought out. Signs belonging to this category are often associated with various more pronounced intra-abdominal morbid conditions recognised by physical examination. It is further not uncommonly of much use to study the effects of deep respiration upon certain of these conditions.

III. INTRA-ABDOMINAL MOVEMENTS.

In some instances the striking abnormal sign in connection with the abdomen observed on inspection is an unusual internal movement ; or such movement may be brought out by palpation or percussion. It may be quite independent, or is associated with other more definite physical changes. The movement is in the large majority of cases associated with the stomach, intestines, or both ; and is either local, or more or less diffused, changing its site, and presenting a characteristic wave-like or vermicular course. A careful study of this sign may be highly suggestive and helpful in diagnosis. Sounds may be brought out during the movement, due to gas and fluid, either audible in the vicinity of the patient or on auscultation. Uterine movements associated with pregnancy or tumour may also be mentioned under this heading.

IV. GENERAL ENLARGEMENTS.

The immediate purpose in a large number of abdominal cases is to study and investigate various conditions giving rise to general enlargement of this region. They are numerous, and, therefore, at the outset it is desirable to have some comprehensive idea of their nature. In infants and young children the abdomen is naturally large in proportion ; while posture may produce an apparent enlargement which does not actually exist, as when a patient suffering from pseudo-hypertrophic muscular paralysis tries to stand erect, the abdomen then protruding forwards in a remarkable degree. Excluding pregnancy, which must never be forgotten in possible cases, the causes of actual general enlargement of the abdomen may be thus grouped :

- I. Corpulence or obesity, the fat accumulating both outside and within the abdominal cavity.

II. Conditions of the abdominal walls :

- (1) Relaxation and distension, often with wasting of tissues.
- (2) Subcutaneous œdema or emphysema.
- (3) An enormous ventral hernia, associated with an opening in the walls.

III. Accumulations of gas internally :

- (1) Within the alimentary canal—Flatulent distension—Tympanites.
- (2) In the peritoneal cavity—Pneumo-peritoneum.

IV. Accumulations of fluid within the abdomen :

- (1) Peritoneal effusion—Ascites.
- (2) Extreme distension of bladder or of pelvis of kidney.
- (3) Cystic accumulation, usually connected with some organ—rarely with the peritoneum, especially hydatid tumour. A pancreatic cyst may originate what is practically a general enlargement of the abdomen.

V. Great dilatation of the stomach or colon, with accumulation of gas, fluid, or both.

VI. An enormous collection of fæces.

VII. Extensive solid infiltration or growth, especially involving the peritoneum :

- (1) Certain cases of chronic peritonitis, especially with extensive adhesion to the abdominal wall, or with much thickening.
- (2) Tubercular disease and its consequences.
- (3) Malignant growths, colloid cancer being particularly important.

VIII. Extreme enlargement of one or more of the solid organs—spleen, liver, or kidney—due to certain diseases, or to the presence of definite growths.

IX. Conditions associated with the female generative organs, especially uterine or ovarian tumours.

X. So-called "Phantom tumour."

XI. Combined conditions. Without entering into any details, it cannot be too strongly insisted upon that general enlargement of the abdomen is frequently due to two or more causes, not only affecting the same structure, as the peritoneum, but involving different parts. The association of peritoneal effusion with excess of gas in the alimentary canal is very common.

COURSE OF EXAMINATION.—I. General enlargements of the abdomen are in the first instance investigated by the ordinary physical methods applied through the walls ; and in order to carry

this out intelligently and practically, it is most desirable to adopt some systematic course of procedure. It is not my intention to describe in this communication the physical signs of the several conditions just enumerated, but merely to indicate the lines to be followed when carrying out physical examination in any case of general enlargement of the region, with a view to diagnosis. Such examination is not always sufficient, and then further steps have to be taken to clear up matters, which will be mentioned later on. The points to be noted and studied in succession are as follows :

(1) The superficial structures and abdominal walls, the condition of these walls often partly or entirely accounting for the enlargement, or affording valuable signs of certain intra-abdominal conditions, as evidenced by the skin, umbilicus, enlarged veins, etc.

(2) The degree of enlargement, which ranges from slight to extreme ; and whether it is liable to more or less rapid variations. Measurements and cyrtometric tracings may be required to give more definite information on these points.

(3) The shape and outline, whether symmetrical or not ; if the abdomen is uniformly rounded, prominent in front, or tending to bulge in dependent parts.

(4) Whether the abdominal respiratory movements are restricted or arrested ; and if any friction-fremitus is produced during these movements in certain cases.

(5) If visible movements of the alimentary canal occur spontaneously, or are brought out by tactile irritation.

(6) The tactile sensations conveyed on palpation and manipulation. It is very important to note these carefully over the whole abdomen, both in connection with superficial conditions, and also as revealing various internal morbid states. In relation to the latter the chief points to determine are the degree of tension of the abdominal walls, and whether this gives the impression of stretching by gas or fluid from within ; smoothness or regularity, or the reverse ; the presence of the elastic or fluctuating feel of a cyst ; various degrees of firmness and resistance, associated with a definite organ or organs, or indicating abnormal solid materials of different kinds, either widely and uniformly distributed, diffused, or localised ; and the development of intra-abdominal movements or gurgling

sensations on manipulation. Although not a physical sign, it is often important to note during palpation whether tenderness is present, as well as its degree, situation, and extent.

(7) Whether fluctuation-wave can be elicited, noting also its seat, extent, and facility of production.

(8) Percussion-signs, including both sounds and sensations. The percussion-sounds are practically either dullness, or various degrees and qualities of hollow sounds, usually of a more or less tympanitic character. They are often associated in the same case, and careful percussion may be required to determine their relative distribution and localisation. In exceptional cases the "bell-sound" can be elicited. The tactile sensations on percussion are also not uncommonly very suggestive and helpful in diagnosis. In certain cases it is requisite to examine specially for hydatid fremitus.

(9) Auscultation-signs, if any. Auscultation is seldom of positive use in the investigation of general enlargements of the abdomen, apart from pregnancy. Possibly friction-sound may be heard; or sounds due to gastric or intestinal movements. Among the rarer auscultatory phenomena may be mentioned remarkable audibility of the aortic second sound over the abdomen, with a metallic quality, associated with pneumo-peritoneum; and a pressure-murmur over either iliac artery. Auscultatory percussion is of use in certain cases.

(10) Succussion-signs, only met with under very exceptional circumstances.

(11) Effects of changes of posture. These are often of great significance, as bringing about modifications of certain signs, especially the shape of the abdomen, percussion-sounds, and fluctuation-wave. The absence of any such changes is in other instances very helpful in diagnosis.

II. Should the results of physical examination thus far described not be sufficient for diagnosis, it may be necessary to study the signs revealed by examination *per rectum* or *per vaginam*. The urine should, of course, always be examined; and if there be any reason to suspect accumulation in the bladder a suitable instrument must be passed. The bowels must be thoroughly emptied if there is any reason to suspect a faecal accumulation.

III. In doubtful or obscure cases it may be still further essential to take into consideration the results of one or more of the special modes of

investigation applicable to the abdomen. It may thus be necessary to examine particular organs systematically; to administer an anæsthetic; to employ skiagraphy; to introduce the aspirator or exploring trochar; or to perform more formidable exploratory operations of different kinds.

IV. Examination of the chest, with the view of determining the presence or absence and nature of thoracic signs, is, in some instances, of much value in the diagnosis of general abdominal enlargements.

V. DEPRESSION OR RETRACTION.

Retraction of the abdomen is at once evident on inspection, and the only points to determine are its extent, localisation, and degree. In most cases it is general, but is sometimes limited to the upper or lower part. In extreme cases the abdominal wall seems almost to touch the spine, the region being "boat-shaped"; while the surrounding bony projections and the thorax stand out with undue prominence. The retracted condition usually enables examination of internal structures to be carried out very easily and effectually, but it is necessary to guard under these circumstances against being misled by exaggerated signs.

Retracted abdomen may be directly due to muscular spasm; emptiness and contraction of the alimentary canal; wasting of structures; or peritoneal adhesions. Its more obvious causes, in addition to temporary retraction associated with disordered respiration, may be thus grouped:

I. General emaciation from any cause.

II. Starvation, both on account of the general wasting, and the empty contracted state of the stomach and intestines.

III. Chronic diarrhoea in certain cases, due to intestinal ulceration or other causes.

IV. Any obstructive disease preventing the entrance of food into the alimentary canal or its onward passage, involving the œsophagus, stomach, or upper parts of the intestine. Pancreatic disease may partly cause retraction in this way.

V. Some forms of intestinal colic, especially lead colic.

VI. Chronic peritonitis in some instances.

VII. Certain cases of disease of the brain or its meninges, especially acute tubercular meningitis.

VI. LOCAL MORBID CONDITIONS.

The more or less localised abnormal conditions affecting the abdominal contents, to be studied by physical and other methods of skilled investigation,

are very numerous and present much variety. I propose to attempt here a comprehensive and practical classification, which may at any rate suggest the main lines upon which the investigation has to be conducted in a particular case, in relation both to individual organs and structures, and to certain independent or special diseases. I submit the following arrangement :

I. External herniæ, usually intestinal or omental or both ; in exceptional instances involving one or more of the solid organs.

II. Conditions definitely connected with one or more of the viscera :

(1) Displacements, simple or with unusual mobility, which may apply to most of the abdominal organs. It may be noted under this head that the testis sometimes remains undescended, and if forgotten might cause a difficulty in diagnosis.

(2) Abnormal fixation.

(3) Conditions of hollow muscular viscera.

(a) Temporary distension or permanent dilatation, with or without hypertrophy of the walls.

(b) Contraction and diminution in size, limited or more or less diffused ; sometimes accompanied with atrophy of the walls.

(c) Abnormal states of orifices, either in the direction of narrowing or constriction, culminating in stenosis or stricture ; or of enlargement and incompetence.

(d) Diseases of their inner surface, as determined by examination of secretions and discharges, or in suitable cases by direct inspection by means of an endoscopic apparatus.

(e) Intussusception, or the protrusion of one portion of a hollow viscus into another portion, practically limited to the intestines.

(f) Accumulations in the interior of a hollow organ, of gases, fluids, or both ; solid materials, it may be in considerable masses, fæcal accumulations being specially borne in mind ; calculi or morbid concretions ; and intestinal worms rarely.

(g) Morbid growth, either as an indefinite localised thickening or firmness ; an evident infiltration more or less extensive ; or a well-defined solid tumour, involving some part of the wall of a viscus, or an orifice

or its vicinity, and occasionally projecting into its interior.

(4) Conditions of solid organs.

(a) Enlargement, varying much in nature and degree, either more or less uniform and regular ; presenting various kinds of firm irregularity, sometimes with definite solid projections ; revealing prominences or special tactile signs of localised accumulations of fluid, chiefly cystic ; or exceptionally exhibiting pulsation.

(b) Contraction and diminution in size.

(c) Changes in shape or physical characters, without obvious alteration in dimensions.

(5) Conditions associated with ducts or tubes.

(a) Obstruction of various kinds, with consequent dilatation behind the point of difficulty, and accumulation of fluid, which in exceptional instances leads to the formation of a considerable swelling, or even a cyst.

(b) Hypertrophy or thickening of the walls, which can be felt in some instances.

(c) Lodgment of a calculus, which may also occasionally be detected by digital examination.

(6) Conditions of the peritoneum and sub-peritoneal tissue ; or of the cellular tissue in certain regions. The great omentum demands special attention in this connection.

(a) Inflammatory exudation in the cellular tissue around certain organs, often ending in the formation of an abscess.

(b) Local peritonitis, which is either dry or attended with effusion, and may also terminate in limited suppuration.

(c) A limited firm thickening, induration, or infiltration, or even a more definite mass, due to chronic peritonitis or new growth.

(d) Local adhesions, leading to abnormal fixation of an organ, or to the matting together of adjacent structures.

(e) Collections of fluid, either in the omentum, or limited by adhesions, or contained in cysts, especially hydatids.

(f) A limited accumulation of gas rarely.

(g) The presence of abnormal materials, more or less solid, in the peritoneal sac, the result of some rupture or perforation, such as fæces, calculi, or foreign bodies.

(7) Special abscesses. Under this head may be included :

(a) Subphrenic, right or left.

(b) Pelvic, originating in the pelvic cavity.

(c) Lumbar, psoas, or iliac abscess—usually due to disease of bone or joint.

(d) Glandular, starting in the absorbent glands.

(8) Special growths or tumours.

(a) Those associated with the intra-abdominal lymphatic or lacteal glands which either present separate enlargement or are aggregated into lumps or masses, which may form considerable tumours. Retro-peritoneal tumour is an important variety coming under this category.

(b) Tumours starting from certain parts of the abdominal or pelvic boundaries, such as the diaphragm, spinal column, or ilium, and encroaching upon the cavity.

(c) Ovarian, uterine, and allied enlargements or tumours of all kinds; not forgetting extra-uterine foetation.

(d) Solid tumours due to infiltration of and around certain organs, especially the bladder and kidney.

(e) Growths projecting from an organ, but only attached to it by a narrow neck or pedicle, so that it may be impossible to make out the connection.

(f) Indefinite growths, involving more than one structure.

(9) Conditions of the larger blood-vessels within the abdomen.

(a) Aneurysm, usually aortic, exceptionally associated with some other artery.

(b) Extensive atheroma or calcification of the aorta, often accompanied with more or less dilatation.

(c) Pulsating aorta, without any organic change.

(d) Obstruction of the inferior vena cava or one of the iliac veins, not recognised by direct examination but by its effects.

COURSE OF INVESTIGATION.—The course of procedure in the investigation of local abdominal morbid conditions must necessarily be modified according to their seat, nature, and other circumstances, but there are certain general rules which

may be followed, and to which attention will now be directed.

1. Inspection cannot in the large majority of cases give any reliable information in this class of case, but any local abnormal signs should be noted, such as changes in the skin; localised oedema or enlargement of veins; a limited fulness or prominence, or retraction; or any evident pulsation. A visible local fulness or enlargement may be very suggestive of certain conditions, or even quite characteristic in exceptional instances. A superficial condition, such as hernia, will probably be obvious at a glance.

2. The abdomen should next be palpated or manipulated in a general way to determine whether anything unusual can be felt; and if there be more than one morbid condition, in different regions, either more or less connected or entirely independent. In the latter case each condition will require separate investigation.

3. In the further procedure it is most important to understand clearly how to examine each organ and system within the abdomen; to conduct such examination on systematic lines, when this is indicated; and to have an intelligent knowledge of the changes from the normal which they may severally present. It not uncommonly happens that the examination thus far reveals at once that we have to deal with some particular viscus or viscera, and the course of investigation required becomes then plain enough.

4. In many instances it is found that there is some definite mass to be felt within the abdomen, and this has to be investigated on certain lines in order to determine whether it is associated with one of the organs or other structures, or is more or less independent, as well as to make out its nature. The points to be noted are :

(a) Its situation, as regards the particular region of the abdomen it occupies; whether it extends into the pelvis; if it passes within the margin of the thorax, the lower part of which it may distend unilaterally, and even modify the intercostal spaces; and its depth in the abdominal cavity, especially observing whether it is obviously superficial, or lies deep down near the spine, or, should it come to the surface, whether it has deep attachments.

(b) The extent or dimensions, so far as this point can be determined by palpation. Should there be any obvious prominence over any part of

the abdomen, local measurements may be of service for the purpose of recording details.

(c) The general shape or outline—paying careful attention to the margins or borders, noting whether they are sharp and well-defined, rounded, or indefinite; as well as to the surfaces. During this part of the examination it is important to recognise any correspondence in shape with that of either of the abdominal viscera.

(d) Whether the surface and borders are smooth and uniform, or present irregularities in the way of diffused granulations, nodular, lobular, or other projections of various sizes and shapes. Sometimes apparent prominences are really due to adjacent depressions, which can perhaps be made out by digital examination; or there may be one or more marginal notches, as in the case of the spleen.

(e) The consistence and other ordinary tactile sensations. The chief varieties in this class of signs met with are dense hardness; more or less firmness, which may be the characteristic feel of a particular organ; softness or a doughy sensation; elasticity; tension; or fluctuation, both digital, and that brought about by filliping. It is sometimes important to determine whether the consistence is uniform or not over the mass under investigation; as well as if any change is produced on manipulation or pressure; or whether any gurgling, grating, or other unusual sensation is thus elicited. The peculiar feel associated with gall-stones in the gall-bladder may be mentioned here, although it is not met with anything like so often as is generally supposed.

(f) Whether the object under examination is absolutely fixed or more or less movable, and in the latter case the degree of mobility on manipulation, and in some conditions in association with the respiratory act. Sometimes a mass is mobile as a whole, but evidently from a fixed point, which may be highly significant.

(g) The presence of any peculiar tactile sensation, such as friction-fremitus; pulsation, which, should it be noted, requires special investigation; or in exceptional cases a thrill. The signs last mentioned may be met with altogether apart from aneurysmal tumours.

(h) Percussion-signs, both sounds and tactile sensations. These are often of the greatest value in determining the precise limits and characters of an intra-abdominal mass; and it is also necessary in certain cases to make out more or less carefully

whether the phenomena are uniform or not over the entire surface, or whether they differ with light and heavy percussion. Hydatid fremitus may occasionally be elicited, and is a valuable sign when present. The fact that an abdominal morbid condition encroaches upon the thorax can be best determined by percussion as a rule.

(i) Auscultation-signs. These are seldom of much help in the investigation of intra-abdominal local conditions, but it may be worth while to note sometimes whether any friction-sound is produced during respiration; and, of course, anything like a murmur or other special auscultatory sign should receive due attention.

(j) Effects of change of posture. These may be highly significant, by modifying the situation of a mass; altering the percussion signs; or influencing fluctuation, pulsation, or murmur, should either of these signs be present.

(k) Phenomena revealed by special methods of examination. These must be carried out as occasion requires, and they need not be recapitulated here. It may again be emphasized, however, that as local collections of fæces are of very common occurrence, care must be taken to empty the bowel in any doubtful case. The valuable help afforded by an anæsthetic in carrying out thorough and efficient local examination of the abdomen is also worthy of note.

5. There are certain intra-abdominal local morbid conditions which do not exactly belong to the last category, and when obviously present or suspected, they require to be examined each on its own particular lines, by physical and other methods. They are chiefly connected with the alimentary canal, namely, dilatation of the stomach or part of the intestine; affections of the gastric orifices apart from tumour; local intestinal obstruction; intussusception; hernia, which may also include other structures; intestinal worms; and a growth or other morbid state involving the rectum. Under this head may also be mentioned aneurysm and other forms of arterial pulsation; gall-stones; renal or vesical calculus; and diseases of organs difficult to reach by physical methods, such as the pancreas and supra-renal bodies. Of course early pregnancy and other conditions associated with the female generative organs are recognised by special methods of investigation or signs.

Another group of cases which may be referred

to in this connection are those presenting indefinite local changes enumerated in the list already given, such as limited thickening or adhesion; dilatation or thickening of a tube; or the lodgment of a calculus in a duct. Such conditions can sometimes be made out by very thorough and careful digital examination through the abdominal walls, or at any rate their existence may be sufficiently probable to warrant the adoption of other methods of investigation, it may be even laparotomy under certain circumstances. An important group of indefinite lesions to be particularly borne in mind at the present day are those connected with the *appendix vermiformis*.

6. It will suffice to mention, as a separate class, those affections which are recognised, not by any physical or objective signs, but by the phenomena revealed by the examination of secretions, excretions, and discharges, more or less elaborate, on the lines already described. Prominent illustrations are afforded by diseases of the kidneys or other parts of the urinary system, the stomach, and the intestines.

VII. COMBINED CONDITIONS.

The fact must never be forgotten when examining the abdomen, that the morbid conditions thus far discussed are often met with in various combinations, which it is most important to recognise from a diagnostic point of view. Without entering into any details, the general nature of such combinations may be thus indicated:

(1) The presence of two or more conditions contributing to general enlargement of the abdomen, a point already referred to.

(2) The association of one or more local diseases with general abdominal enlargement or retraction.

(3) Combinations of local morbid changes in great variety, either affecting the same organ; involving two or more viscera; implicating the peritoneum as well as other structures; leading to accumulations of fæces or fluids; or being entirely independent and distinct.

The course of procedure in the investigation of cases coming under this category must be guided by circumstances, the examination being conducted on the lines just discussed, which an intelligent and experienced clinical observer will be able to apply and modify accordingly.

REMARKS ON THE TREATMENT OF THE IMPERFECTLY DESCENDED TESTICLE AND ITS DISEASES.

Delivered before the Medical Society of
University College Hospital.

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To the anatomist, comparative anatomist, and embryologist, as well as to the practical surgeon, the imperfectly descended testicle presents many problems of the greatest interest.

It is, indeed, difficult to say from which special scientific standpoint the subject proves most attractive, and were it possible this evening to consider it from them all, you would find each one revealing its own special features of interest; and the smaller details of one science would become both attractive and important when viewed in the light of another.

But the time at our disposal will not permit of such a comprehensive survey; and I propose, therefore, to deal with the imperfectly descended testicle from the surgical point of view alone. This, I think, will be the most interesting, and useful to you; and I venture to bring the subject before you, as the deformity is a fairly common one, with special complications of its own, concerning which you will find but little information in the ordinary text-books on surgery.

We will first consider the various forms of imperfect descent, and the difficulties in diagnosis they may present:

A. PARTIAL DESCENT, in which the testicle has taken up a permanent position at some point along the line of normal descent. This form may be divided according to the situation of the testicle into:

(1) *Abdominal*, when the testicle remains within the abdomen;

(2) *Inguinal*, when the testicle lies in the canal, or about the inguinal region;

(3) *Scrotal*, when the testicle lies in the upper part of the scrotum.

B. ECTOPIC DESCENT, by which is meant a descent to some abnormal position, outside the line of ordinary foetal descent. This variety is much more rare, and the position assumed by the testicle may be one of the following, in order of frequency:

(1) *Perineal*, in which the gland lies beneath the skin of the perinæum, just in front of the anus, in the normal condition of the testicle of the pig.

(2) *Scroto-femoral*, in which the testicle has stopped short of the perinæum, and come to lie in the scroto-femoral groove.

(3) *Femoral*, in which the testicle has descended through the external ring, and passed beneath the skin of Scarpa's triangle, instead of that of the scrotum.

(4) *Penile*, in which the testis rests beneath the skin covering the dorsum penis.

In the abdominal variety the testicle may remain in its early foetal position, just below the kidney; but this is extremely rare; and in almost all cases the gland has been found in the iliac fossa, where, indeed, it may often be felt on deep palpation.

The inguinal region is by far the most common site of the imperfectly descended testicle, and the organ may lie high up, or low down, in the inguinal canal; or, having traversed the latter, it may remain at some point beneath the skin, in the neighbourhood of the external abdominal ring.

But such slight differences in position are of minor interest only, and it is of greater practical importance to remember that the testicle is small, and soft, often difficult to feel, and usually very movable. This last quality, depending as it does on the long mesorchis and large vaginal sac which such testes possess, is worthy of notice; for it allows the testicle to slip up and down the inguinal canal, so that it may resemble a hernia; and, moreover, it is in consequence of this same anatomical peculiarity that the testicle may become rotated on its pedicle, or extruded through the external ring; whereby it becomes strangulated in a peculiar manner, as will be seen later.

The resemblance to an inguinal bubocele is often very great, for the tumour may apparently reduce and disappear on lying down, coming down again on standing, and possessing a marked impulse on coughing; all due to the mobility of the testicle in the inguinal canal.

Many such cases are wrongly diagnosed as ruptures, and the patient is enjoined to wear a truss.

Now this is wrong, and especially so in a young subject, for it prevents all possibility of a subsequent descent, which otherwise might occur. Moreover, the pressure of the truss tends to destroy

the testicle, and may cause the patient considerable pain.

You must have seen several cases among the out-patients where such a mistake has been made and a truss applied to the supposed rupture, and the patient has come to hospital complaining of the pain caused by it. It is obviously easy to avoid this error if an examination of the scrotum be made and both testicles felt for; and this should invariably be done in all cases of supposed rupture or other tumour about the region of the scrotum, especially in infants, in whom the parts are hardest to define, and to whom the application of a truss will do most harm.

The ectopic varieties of testicle are also liable to faulty diagnosis and treatment, if the possibility of their occurrence be not borne in mind, and an examination of the scrotum made.

We will now turn to consider what effects imperfect descent produces on the testicle, and its functions; and what diseases are liable to be set up in consequence of the malposition of the gland. We shall then be in a position to estimate the dangers that are run if the testicle be left alone; and to discuss which may be the best line of treatment to adopt in order to avoid them.

And first to consider the effects of imperfect descent on the testicle and its functions:

The normal testicle possesses two functions—

(1) An influence over the growth of the body, most marked at puberty, imparting to it its masculine characteristics.

(2) The spermatogenic function developing at puberty, and marked at this period by rapid changes in the tubules of the testis, which increase greatly in size, and acquire a lumen surrounded by cells showing the various phases preceding the production of spermatozoa.

In cases of imperfect descent, the first of these two functions could only show signs of being interfered with should both organs be affected, and even in such cases it is certainly most uncommon for the patient to display any departure from bodily growth suggestive of an eunuchoid condition.

No interference with the testicle, therefore, is called for on this account, for even in the case of a bilateral cryptorchid both body and mind may be expected to develop and display the distinctive characteristics of the male.

As regards the second function, however, the opposite effect obtains, and as a sperm-producing organ the imperfectly descended testicle may always be regarded as useless.

The proof of this statement rests partly on the absence of spermatozoa from the seminal discharges in bilateral cases, and from the vesiculæ seminales of the affected side, where one testicle only is affected; and chiefly on an examination of the microscopic sections cut from a number of imperfectly descended testicles from patients of various ages.

Such sections show two main changes—

(1) A general fibrosis throughout the body of the testicle, starting at a very early age and progressively increasing, which interferes with the nutrition of the cellular elements of the tubules, preventing their development, and causing them to atrophy.

(2) An almost total absence of that marked development of the tubules at puberty which is the necessary preliminary to the production of spermatozoa.

There is no evidence of any fault in the embryonic development of such testes, as their structure immediately after birth is similar to that of normally situated organs, and there is no question as to the patency of the vas and the presence of a properly developed spermatic artery.

Therefore such changes as are found must be caused by—

(1) A want of development directly due to non-descent.

(2) An arrest of development, resulting from the malnutrition caused by the fibrous changes which ensue, in consequence of the injuries sustained by the organ in its faulty position.

And I think that a study of the subject from the point of view of actual practice would lead you to overlook the first-named cause and to emphasise the second, in consequence of the early and irreparable damage to the testicle wrought by the latter. The former, however, has certainly good grounds for existence, in view of the analogy, as shown by Griffith, between the imperfectly descended testicle and the non-active testicle of rutting animals, whereby the failure to descend, and some of the want of development found in the testicle, may be ascribed to a reversion to a more primitive type.

The sections which you see here before you sufficiently illustrate the chief changes found.

Those removed before the age of puberty show, on comparison with healthy testes at the same age, only an intertubular fibrosis, of greater or less severity, with some atrophy of the tubules. The cells of the tubules are comparable in every way with those of health, and in the specimen removed from an infant eighteen months of age it would really be difficult to note any departure from the normal in them.

At puberty, you notice how the healthy tubule increases in size, and acquires a lumen, round which the cells become ranged in several layers, showing all the extraordinary changes preceding the formation of spermatozoa.

Such rapid growth and functional activity demand a concomitant increase in blood-supply, and such an increase is impossible in the fibrosed testicle of imperfect descent, as you may see in the specimen before you. This testicle was removed from a boy aged eight, who had worn a truss, and in it you note a great deal of newly-formed dense fibrous tissue, with flattening and atrophy of the tubules and vessels, quite sufficient to prevent its subsequent development.

In the imperfectly-descended testicle at puberty, therefore, the tubules either undergo no development, or, having partially developed, and as it were threatened spermatogenesis, their growth is checked by the advancing fibrosis, and they rapidly commence to undergo fatty degeneration and atrophy.

Such partially developed tubules, showing marked degeneration and unequal growth, you can see well in this section from an inguinal testicle of a youth of 18; and some of its tubules are lined by a single layer of columnar cells with ragged extremities, such as Griffith has shown to exist in the non-active testes of rutting animals. Sections from older subjects only show more advanced degeneration and fibrosis, still further diminishing the possibility of spermatogenesis, and shortly it may be said that in no section from an imperfectly descended testicle has a well-developed spermatozoon ever been seen.

It is evident, therefore, that the testicle must be removed from its abnormal position if its function is to be preserved; and since advanced stages may be found at a very early age, there should be no delay in replacing the organ in the scrotum.

We will now, however, turn and consider the diseases that may occur in consequence of imperfect descent, and see what part they play in influencing the treatment of such testicles.

Included under this heading are: (1) Hernia; (2) inflammatory diseases; (3) strangulation; (4) tumours.

By far the most frequent is hernia, for it is found in about 10 per cent. of all cases of imperfect descent.

The rupture may develop at an early age; but as a rule it appears soon after birth; and in order to cure it an operation is necessary, or a truss may be put on, which will keep up both the rupture and the testicle. The latter alternative is by no means to be desired, for the possibility of a late descent is thereby prevented. Moreover, the testicle is often pressed on by the truss, and rapidly atrophies, and in some cases the compression gives rise to so much pain that the truss cannot be worn.

In seeking for the causes that so frequently lead to hernia in this condition, we find them in the peculiar disposition of the serous covering of the testicle in these cases.

The patent processus vaginalis testis should, as you know, become closed above immediately after birth, cutting off the tunica vaginalis from the peritoneum. In imperfect descent this phase of development is wanting in 67 per cent. of all cases, and the processus vaginalis remains in its foetal condition, communicating freely with the peritoneum above, and offering free passage to any loop of bowel that may be forced into it.

There are some other peculiarities in connection with the herniæ accompanying imperfect descent which are occasionally seen, and which seem to depend on the peculiar shape of, or course pursued by, the processus vaginalis in these cases. Though rarely seen, they are worthy of mention on account of the difficulty in diagnosis that may arise should strangulation occur.

Thus the hernial sac may be found reaching into the thigh or perinæum, while the testis lies in the inguinal canal, or abdomen; a condition of affairs which seems to be due to an abnormal activity of those fibres of the gubernaculum which draw down the processus vaginalis in front of the testicle.

In other cases, again, the hernial sac extends upwards and outwards between the muscles of the

abdominal wall, giving rise to one of the forms of interstitial hernia; or a pouch of the sac running up in the subperitoneal fat of the iliac fossa or abdominal wall, about the region of the internal ring, produces a pro-peritoneal hernia.

In seventy per cent. of these forms of hernia the testicle is imperfectly descended, and the frequent connection of the two deformities, whilst indicating a common ætiology, may serve also as a guide in the diagnosis of the hernia; and for this reason it is worth remembering, on account of the difficulty in diagnosis which may occur when such herniæ become strangulated; and this, especially with the pro-peritoneal variety, in which the local signs of hernia may be almost or entirely wanting. The onset of symptoms of acute obstruction in a patient with an imperfectly descended testicle should always bring to your mind, therefore, the possibility of this condition, even though there be no local signs of strangulated hernia present.

The following general conclusions may be therefore drawn with regard to hernia accompanying imperfect descent:

(1) That the imperfectly-descended testicle is frequently complicated by the presence of an accompanying hernia.

(2) That in the treatment of such a hernia a truss either cannot, or ought not, to be worn.

(3) That such a hernia is liable to undergo increase in size and strangulation if left untreated.

(4) That the presence of such a hernia is therefore an indication for operation for the cure of the hernia, in the performance of which the testicle may also be dealt with in one of several ways, to be considered later.

Inflammatory diseases.—The displaced testicle is, in most cases, much more liable to become the recipient of blows and injurious pressure than the normally situated organ.

Hence attacks of inflammation of varying severity are common in such testes, leading to fixation to surrounding parts, and augmenting the fibrotic changes which prevent their development, and ultimately destroy their structure.

In such attacks the testicle becomes swollen, tender, and painful, and the patient often vomits.

If the testicle lie about the inguinal region, a superficial resemblance to strangulated hernia may be set up; for, with the history of a previously reducible tumour, which has become fixed and

tender, and the presence of vomiting, it is possible to be led astray.

Nor, indeed, does the resemblance always stop here; for the concomitant acute hydrocele in the patent processus vaginalis may spread to the lower abdominal peritoneum, causing fever, fixation of the lower abdomen, and constipation, and prolonging the vomiting.

With such a clinical picture, one's impressions in favour of strangulated hernia may be strong, though erroneous, and, on the contrary, it may not be enough to recognise the absence of the testicle from the scrotum and ascribe all the symptoms to its having become inflamed; for with the testicle there may, indeed, be a hernia in many cases, as we have seen, and this, though small, may have become strangulated, giving rise to the group of symptoms under consideration.

Whilst it would suffice, therefore, to apply fomentations only in a clear case of orchitis, even with secondary peritonitis, in all cases of doubt it would be wiser to explore the condition of affairs present by means of operation, lest a strangulated hernia should also exist.

The comparative frequency of orchitis as a result of injury in the imperfectly descended testicle makes the rarity with which gonorrhoeal orchitis is met with in the same condition all the more striking.

Affection with tubercular disease is also extremely rare, and it is probable that the immunity from both these diseases depends on the small size and empty condition of the vas and vesiculæ, in consequence of which the usual channel of infection is almost non-existent.

Strangulation.—Strangulation of the testicle is a disease almost wholly confined to imperfectly descended testes; and it is of interest both on this account, and also because of the difficulty in diagnosis which has led, in most cases, to the condition being mistaken for strangulated hernia.

The pathology of the process is, on the whole, similar to that which takes place in a strangulated loop of gut or twisted ovarian tumour; the vessels of the cord become pressed on, and the testicle becomes intensely congested, and subsequently necroses.

The cause of this condition is, in most cases, a torsion of the cord, permitted, apparently, by the long mesentery or mesorchis, which is a marked

feature of the imperfectly descended (as of the foetal) testicle. The whole testicle becomes rotated on the mesorchis, acting as a pedicle; and the latter becomes screwed up so tightly that the vessels of the cord are pressed on and obliterated. In some cases, however, the process appears to be due more to tension than torsion; a testicle normally lying in the inguinal canal being forcibly extruded through the external ring. The cord is so stretched that the vessels are pressed on, and strangulation ensues.

The latter explanation, which was the only one given before torsion was discovered by Nicoladoni, has been more or less disregarded since. But I am none the less sure that it actually occurs, as I have seen such a case, which at subsequent operation showed no signs of torsion of the cord. The symptoms in this case could be brought on at once by forcing the testicle down through the external ring, the pain continuing until it was "reduced."

The symptoms of strangulation may develop gradually, or suddenly, often after some blow or strain.

The testicle becomes painful and tender, and soon swells up beyond recognition; and in those cases in which gangrene has occurred, the overlying skin has become both dusky and inflamed.

If seen within an hour or two of the onset, it may be possible to recognise the inverted position of the testicle and the knot above it formed by the twist in the cord. In two such cases the twist has been successfully reduced by manipulation. But when seen later, a painful swelling in the inguinal region is all that can be definitely detected.

Vomiting usually occurs once or twice at the beginning of strangulation, but in some cases nausea only may be present. Should, however, the consequent inflammation spread, *viâ* the patent processus vaginalis, to the general peritoneum, the vomiting continues, and is accompanied by abdominal tenderness and distension, and more or less complete constipation.

Strangulated hernia is thus strongly simulated both by the previous history of an apparently reducible tumour, and the onset of acute symptoms after a strain or blow. The general combination of symptoms and signs present is also such as would lead to this mistake. But the obstruction is rarely complete, nor are the other symptoms so severe as in an ordinary strangulated hernia.

It is well, however, to bear in mind that such a group of symptoms may be due to several pathological conditions, including an inflamed or strangulated imperfectly descended testis, strangulated omentum, vermiform appendix, or Meckel's diverticulum; and strangulated, partial enterocele, or Richter's hernia.

In all cases of strangulation of the testicle in which the symptoms cannot be reduced by manipulation, it will be necessary to make an exploratory incision, and, in most cases, to remove the gland; for unless the strangulation be very mild, either gangrene or rapid atrophy will ensue if the cord be merely untwisted and the testicle replaced.

Tumours.—The last question to be considered is the one of tumours affecting the imperfectly descended testicle. These may be either sarcomata or carcinomata, and they may affect the testicle in any form of imperfect descent. It has usually been stated that imperfectly descended testes were more liable to become the seat of malignant disease than those normally situated; but the number of recorded cases would not lead one to this conclusion, nor does a comparison of the malignant tumours of the testicle seen during the last ten years in University College Hospital tend to show that such is the case. The peculiarity of such tumours, rather than their relative frequency, would appear to have emphasised them in the minds of observers, and gained them a place of greater note in surgical literature than they numerically deserve. When affecting testes within the abdomen, such tumours form oval movable masses, with a definite pedicle, formed by the mesorchis, which isolates them, and renders their removal quite simple if they be recognised before the new growth has begun to infiltrate surrounding parts. In the groin they more quickly become adherent and fixed, and surrounding parts are soon affected, rendering removal difficult, or even impossible. There is very little difference to the naked eye between either form of malignant disease, and by early diagnosis and complete removal only can they be successfully treated. Any peculiar rounded tumour in the groin or iliac fossa should at once lead to an examination of the scrotum; and if the testicle be absent, the origin of such tumour is at least strongly indicated.

If left to grow, the mass becomes fixed to the

iliac vessels and intestines, and secondary deposits form tumours in the lumbar glands.

Treatment.—Our survey, so far, of the undescended testicle and its diseases, leads us to the following conclusions:

(1) That in order to preserve the spermatogenic function of the gland it must be removed from its abnormal position as early as possible.

(2) That none of the secondary diseases to which it is liable are sufficiently severe or frequent to justify operative interference as a preventive measure.

(3) That all cases of hernia must be treated by operation, as must all cases of unreduced strangulation of the testicle, all tumours, and doubtful inflammatory swellings.

We are left, therefore, with two alternatives with regard to the treatment of the testicle. If it can be made to grow by bringing it down into the scrotum, then such reduction or transplantation should be performed at once. If such a procedure prove futile as regards future growth, and the development of the spermatogenic function, then the testicle is better left alone; unless the onset of pain, or some concomitant disorder, such as hernia, call for operation, when the useless gland may be removed.

Three modes of procedure have been advocated with a view to preserving the functions of the gland:

(1) To bring down the testicle into the scrotum by manipulation.

(2) To replace the testicle into the abdominal cavity, and so preserve it from injury.

(3) To bring down the testicle to the bottom of the scrotum by operation and fix it there.

Each of these methods may now be reviewed.

The first one, in which the testicle is brought down by manipulation, is certainly sufficiently successful if practised early, and no doubt you have seen such cases successfully treated among the children brought as out-patients to the hospital.

If the mother can be made to press down the testicle towards the scrotum several times daily, and hold it there for a few minutes, the chances of success in a child under two years of age are fairly good. At a later age the prospects are by no means so bright, and as the gland is beginning to undergo fibrosis, too much time should not be

wasted in needless attempts to draw it down by this method.

Reposition of the testicle within the abdomen has nothing to recommend it, for it is well known now that the abdominal testicle is as little liable to undergo full development as its fellow in the groin—a fact which lends some support to the theory of non-development being partly due *ipso facto* to non-descent.

The third proposition—to transplant the testicle to the scrotum by operation—is one that has been

sufficient length of time to prove that the testicle actually did increase in size, and remain permanently in good position. For in many cases after a few months, the gland tends to return to its former situation, and an incipient increase in size may not be maintained. In order, therefore, that we may judge of such results, it is necessary to follow up a series of cases of orchidopexy, and note the condition of the testicle some years after the operation.

The table of thirteen consecutive cases* will give you some idea of the poor results obtained.

No.	Age in years	Position of testis before operation	Form of operation	How long after operation examined	Size of testicle before operation	Size of testicle after operation	Subsequent position of testicle
1	8	At external ring	Wire cage	2 years	Big as fellow	Two thirds of fellow	Just below external ring.
2	16	Inguinal canal	Fixed by suture to adductor tendon	4 years	Slightly smaller than fellow	Half of fellow	Fixed to pubes.
3	17	Inguinal canal	Wire cage	2 years	Slightly smaller than fellow	Size of small bean	Over external ring.
4	16	Inguinal canal	Fixed by suture to adductor tendon	3 years	Slightly smaller	Half of fellow	At external ring.
5	13	Inguinal canal	Wire cage	5 years	Small	One third of fellow	One month after operation halfway down scrotum; five years later over external ring.
6	15	Inguinal canal	Wire cage	3 years	Small	Size of small bean	One month after operation in bottom of scrotum; three years later over external ring.
7	14	Inguinal canal	Wire cage	6 years	Small	Size of pea	One month after operation in top of scrotum; six years later over external ring.
8	3	Inguinal canal	Tied to roll of gauze	7 years	Small	Half size of fellow	In inguinal canal.
9	4	Inguinal canal	Vessels cut, cage	5 years	Very small	Size of pea	Over external ring.
10	15	Over external oblique aponeurosis	Wire cage	2 years	Small	Half size of fellow	One month after operation at fundus of scrotum; halfway up latter two years later.
11	13	Inguinal canal	Wire cage	2 years	Small, cord long	Two thirds size of fellow	In scrotum.
12	18	On external oblique aponeurosis	Wire cage	4 years	Good size	Two thirds of fellow	In scrotum.
13	8	Over external ring	Wire cage	7 years	Rather small	Full size	In scrotum.

widely carried out, and several methods have been adopted to effect this end.

A study of the recorded cases would lead one to believe that such an operation is nearly always possible, and that the results with regard to the testicle are nearly always good.

But there is a tendency for good results only to find their way into literature, and moreover, many of the cases so recorded were not followed up for a

There is, as you will observe, only one case in which the testicle regained its proper size, and in four cases only was the testicle found in the scrotum on examination.

The causes of failure are, I think, chiefly two:

(1) That the operation has been performed too late, when the structures of the cord are shorter.

* Followed for a period of from two to seven years after operation.

and the proper elements of the testicle destroyed, or their development prevented, as the sections show us, by fibrosis.

(2) That an attempt has been made to preserve some testicles which, from the shortening of the vessels of the cord or other gross developmental defects, had better have been removed.

Summary of Treatment.—To sum up the treatment of the imperfectly descended testicle, therefore, it may be said :

(1) That manipulation should be attempted in infants, and regularly carried on for some time.

(2) That should it fail, or should the patient be more than two years of age, recourse should be had at once to operation.

(3) That no attempt should be made to save the testicle if the body and epididymis be widely separated, or if it be necessary to sever all the vessels of the cord in order to bring the gland into the scrotum.

(4) That no operation except castration should be performed if the patient be past the age of puberty. After ten years of age, orchidopexy should not as a rule be attempted, unless an operation exposing the testicle is called for on account of hernia or strangulation, as the testicle is by this time so fibrosed, as a rule, that the chances of subsequent development are too slight to justify an operation with a view to conservation of function alone.

In those cases in which the testicle lies completely concealed in the abdominal cavity, an exploratory operation with a view to finding it, and drawing it into the scrotum, is but little likely to be attended with success, and should only be attempted with a view to establishing the spermatogenic function in a bilateral cryptorchid before puberty.

With regard to the choice of methods of operating, that by which the lower pole of the testicle is drawn down by a suture to Watson Cheyne's wire cage is certainly both effective and convenient. But the testicle should also be fixed by a permanent buried suture to the bottom of the scrotum, and the fascia about the cord, after drawing down the testis to the full, should be sutured to the pillars of the external ring, and the tissues about the root of the scrotum. The chances of subsequent retraction will thus be diminished, and the testicle lying in the fundus of the scrotum will be more likely to develop.

WITH MR. CANTLIE AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

23rd OCTOBER, 1901.

THIS seems to be a hip case, but it is one in which the history is not easily ascertained, because the father and mother are not here, the child having been brought by a friend with whom he has been living. We are told that he did not walk until he was three years of age, and he is now five.

Is it a congenital ailment of the left hip? There seems to be no spinal curvature. The right leg appears to be longer than the left. There is not much difference to be detected in the shape and size of the two limbs. The child walks with a decided limp, "a falling over" towards the left side, as if the muscles of that hip were not sufficiently developed. Sometimes this variety of trouble when no physical lesion is apparent is associated with phymosis, but there is no complication of that kind in this case. If there is no spinal curvature, no phymosis, and no actual difference in the growth and development of the two limbs, the fault may lie in arrested development of one side of the pelvis. If none of these conditions obtain—which they do not in this child,—infantile paralysis suggests itself, but paralysis of one of the muscles of the hip without evidence of paralysis of any part of the limit below, is a very rare disease.

May this be due to dislocation? Nelaton's line shows the great trochanter to encroach considerably in this line, and it is evident that there is displacement of the great trochanter upwards. Displacement upwards may arise from fracture of the neck of the femur, from dislocation, from the head of the femur slipping upwards owing to deficiency of the upper part of the acetabulum, or from the upper part of the acetabulum being chipped off. In examining a hip-joint, for instance, after a fall on the hip, there are one or two points which are of great practical value to bear in mind. A man falls from a height and injures his hip. Three things may happen: he can bruise his hip, or he can have a simple fracture of the neck of his femur, or an impacted fracture. How do you diagnose the one from the other? If it is merely a bruised hip, if you ask the man to raise his hip he will declare he cannot do so; that he is so hurt it

is impossible for him to do it ; and he will say he is sure that his bone is broken. But if you persist in your request that he tries to move his injured limb, you will find—it may be after long beseeching—that he can raise his foot even to a very small extent—say an inch—off the couch, then the bone is not broken.

But if you were dealing with an impacted fracture, how can you distinguish between that and a bruised hip or a simple fracture of the neck of the femur ? In an impacted fracture the patient has still some power in his hip-joint, so that if you persist in asking the injured man to lift his limb off the couch, he will not be able to do so, but, instead, he will drag his heel along the couch, although he cannot raise his heel off the bed. In all cases of accident in the neighbourhood of the hip it is well to make out these points. If it is a bruised hip he can raise it. If it be an impacted fracture he cannot raise his leg, but he can drag his heel along the couch. If it is a simple fracture of the neck of the femur, such as occurs in old people, he will say, "Of course I can move my leg ; it is ridiculous for you to suggest that I have broken my thigh ; I only caught my foot and twisted my leg ; I cannot have broken my bone ; I did not even fall down." There is no difficulty in getting a person with an injury of this nature to try to raise his leg, but movement of any sort is quite impossible. In this case the head, neck, and shaft of the femur seem displaced upwards ; the head of the bone is neither backwards nor forwards ; one cannot feel the head of the femur anywhere, because it is evidently properly sunk in a socket, although above its normal position. What then is the condition ? The upper margin of the acetabulum may have been chipped off, and the head becoming displaced upwards, a new joint has been formed, accounting for the shortening and the great trochanter being moved upwards above the Nelaton's line. This conclusion seems justifiable, as the head of the bone cannot be felt in any position. What can be done ? This boy has a good useful limb, and he can walk about all right. All that seems to me to be required is a thickening of the sole of the left foot for about one third of an inch. This, of course, will not alter the hip lesion, but it will give the child a useful limb.

The question of incipient hip disease arises. But one does not get hip-joint disease at an earlier age

than two and a half years, and this child seems to have been afflicted before that age. It is about that period of life that, owing to a tubercular deposit in the neck of the femur, what is termed femoral coxxyalgia commences. From that—two and a half to five years of age—is the time of life at which this variety of hip-joint occurs. The tubercular deposit in the neck and head of the bone invariably goes on to caries and suppuration, the pus finding its way to the surface by pointing two inches below the great trochanter.

Another form of hip-joint disease consists of necrosis of the bottom of the acetabular cavity. This form of hip ailment commences at a later period of life, namely, between the ages of five and ten years, and is termed acetabular coxxyalgia. Suppuration results, the pus finds its way towards the pelvis ; but its passage thence is barred by the internal obturator muscle and fascia, and the pus travels upwards, and points in the neighbourhood of the groin. A third variety of hip-joint disease commences in the synovial membrane, and its onset is usually deferred to between the ages of ten and fifteen. This is the form which occurs in young adults, and constitutes the arthritic coxxyalgia. It does not necessarily proceed to suppuration.

Thus we get varieties according to the age of the child, but nothing before two and a half years. In the earlier form the disease runs a very rapid course, the pus pointing below the great trochanter. After five years of age there is necrosis, and the pus finds its way towards the cavity of the pelvis, or it may point anywhere around the joint. In the arthritic variety ankylosis is the usual result, but even this form of hip-joint disease may end in suppuration, and if it does pus will reach the surface anywhere round about the joint. This patient has not got hip-joint disease as we understand it. It does not seem to be a case of congenital dislocation, but a displacement upwards of the whole femur, owing to the upper part of the acetabulum being chipped off, or it may be that there has been arrested development in the part of the acetabular cavity formed by the ilium. I do not see that any operation would benefit the patient, and therefore I shall merely recommend that a thickened sole be added to the left boot.

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A CLINICAL LECTURE

ON

RETRO-UTERINE SWELLINGS.

Delivered at Charing Cross Hospital, June 18th, 1901.

By AMAND ROUTH, M.D., B.S., F.R.C.P.

GENTLEMEN,—We have had a good number of cases in the wards lately of retro-uterine swellings, and I was asking my class the other day what subject I should choose for this lecture, and some suggested this subject, with more especial reference to tubal swellings, such as pyosalpinx. There is no doubt about the importance of a subject of this sort.

The commonest position, I suppose, of any defined induration about the pelvis is behind the vertical level of the uterus, and this is a very fit subject to take. As a rule any swelling in Douglas's pouch or behind the uterus and broad ligaments produces a sensation of fulness. To all intents and purposes it is a foreign body lying between the uterus in front and the rectum behind, and bounded laterally by the broad ligaments, which contain the Fallopian tubes and ovaries. So you can see that any swelling there, fixed or mobile, would necessarily cause inconvenience. Therefore when a patient has such a swelling, with or without local inflammation, she has the sense of something in the pelvis which produces pressure, bearing down, backache, interference with the rectum perhaps, and very likely some interference with the function of the uterus, that is to say with menstruation. If you think of the anatomical position of things in the pelvis you will see more particularly how any tumour or lump in the pelvis is likely to be inconvenient to the patient. You have first of all the uterus covered by peritoneum, a tender structure. That peritoneum runs down along the back of the vagina and up again over the rectum, and then is transferred to the front of the pelvic walls. And the broad ligaments, also covered by peritoneum, contain those very tender structures the ovaries and

tubes, and posteriorly there are the blood-vessels running along at the back of the pelvis, with a good many of the nerves, too,—the sacral plexus, and so on. So if there is anything at all in the retro-uterine half of the pelvis, or in one or other posterior quarter of the pelvis, it is extremely likely to cause inconvenience to some of these organs; and even if the patient does not feel actual pain, she gets reflex disturbances carried on through the sympathetic nervous system.

It is a good plan to divide swellings in the pelvis into three main groups: (1) those which are fixed; (2) those which are impacted; and (3) those which are mobile.

With regard to the fixed group, anything which is accompanied with inflammatory exudation becomes fixed. A pelvic peritonitis, or perimetritis as it is called, is perhaps one of the commonest conditions which may fix structures in the pelvis, and that may be secondary to salpingitis, or it may spread from some other organs, such as from an appendix, in the neighbourhood. Then inflammation may be of the cellular tissue, parametritis, which equally fixes any structures, such as the uterus, which is surrounded by this inflamed cellular tissue. These inflammatory exudations may be lymph, or may be serum, as you saw in a case the other day where I opened Douglas's pouch by posterior colpotomy, or it may be pus either encysted by lymph in the peritoneal cavity or altogether subperitoneal. Instead of exudation you may have infiltration. You will probably remember the patient last week who had carcinoma, and whose disease we decided was too far advanced for us to do anything. In her case the cervix was fixed entirely on one side, and it was doubtful whether it was inflammatory exudation travelling along the broad ligament and the left utero-sacral fold, or whether it was a malignant infiltration. When we came to strip off the bladder in front we found that the induration was continuous with a very friable mass in the left side of the supra-vaginal cervix; that it was, in fact, a case of malignant infiltration. That is what we find in advanced cases of cancer of the cervix, or even cancer of the body of the uterus. A growth spreads along the plane of tissue, cellular or epithelial, and the parts become fixed. One sees that in cases where papilloma of a hilum or broad ligament cyst spreads through the cyst-wall and invades the surrounding tissues. It then becomes

practically a malignant papilloma. Again, we may have hæmorrhages causing fixation. There may be a retro-uterine hæmatocele, the hæmorrhage filling up Douglas's pouch and spreading to the posterior quarters of the pelvis, bounded laterally therefore by the broad ligaments each side, and reaching up and covering, perhaps, the uterus, bounded upwards by adherent matted bowel. That is an encysted collection of blood commonly called retro-uterine hæmatocele, generally the result of a ruptured tubal gestation or tubal abortion. To a less extent fixation may be caused by a broad ligament hæmatoma, which is called an intra-ligamentous hæmatoma. That will also fix the uterus, but fixing it on one side only, or, at all events, on the two sides in different degrees.

There are other fixed tumours in the pelvis, which grow from fixed parts. For instance, there may be osteoma or enchondroma or osteo-sarcoma growing from the front of the sacrum or from the sacro-iliac synchondrosis.

Then there are certain impacted tumours of the pelvis. They are not fixed because you can very often move or replace them under anæsthesia. The most typical of such swellings would be an impacted retroverted gravid uterus, a fairly common condition. Or it might be a fibroid which has grown below the level of the sacral prominence and has become impacted in the pelvis. More rarely a cyst spreading from the ovary may get impacted in the pelvis. But cysts are semi-fluid, and, as a rule, they just rise up out of the pelvis when they get too large to be comfortable there, unless they are fixed by inflammatory exudation, and then that does not apply to our classification, as they are not impacted. Sometimes masses of fæces get impacted in the pelvis, so hard that they feel as if they are not fæcal matter at all. There ought to be no difficulty in determining a fæcal impaction, but, as a matter of fact, there is difficulty. They do not always give that pitting on pressure which you read of in books; and, as other tumours which are impacted are sometimes œdematous, such as a retroverted gravid uterus, mere pitting is not necessarily a proof that it is fæcal matter, so one often finds mistakes have been made in that direction.

With regard to mobile tumours, there are a large number of these, but you must remember that these are only mobile if they are not surrounded

by exudation, or are not a seat of advanced malignant disease. Any one of these mobile tumours may be fixed by one or other condition of this sort. The commonest mobile swelling in the pouch of Douglas is the fundus uteri in the case of retroversion or retro-flexion of the uterus, and that fundus may or may not be the seat of a fibroid nodule. Or it may have a pedunculated fibroid growing from it. So, in addition to the fundus being itself retroverted, a fibroid growing from the fundus may be the cause. Then, again, the swelling may be simply a prolapsed ovary. We often see in retroversion of the uterus that the ovaries are prolapsed also, but apart from that, the ovary may prolapse. Cysts of the ovary almost always get behind the uterus, and as they get heavier and sag down into the pelvis, they take a position which their attachment to the cornu of the uterus necessitates, getting eventually into the sacral hollow. This happens also to dermoids and to anything which is not too large to occupy the sacral hollow.

Now a few words with regard to tubal diseases. Any enlargement of the Fallopian tubes causes the tube to behave exactly as the ovary would do; it sags down from increase of weight, unless fixed by adhesions. The broad ligament is not able to support it, and it tends to get into the middle line, where the pouch of Douglas comes down. Such tubal swelling may be an ordinary inflamed tube from chronic salpingitis, such as we get, for instance, in this specimen. You see a tube laid open, which probably did not contain pus at all, but was lined by very swollen mucous membrane. You will see that the mucous membrane lies in longitudinal folds and is very much hypertrophied. When you feel such a tube in position, after opening the abdomen, you feel sure there is pus inside; but when you make an incision you find there is nothing but swollen mucous membrane, and yet it forms a definite tumour. This specimen is an instance of a swollen tube due to hæmorrhage into it, not as a result of a tubal mole, or a tubal gestation at all, but apparently a hæmorrhage apart from that cause. No chorionic villi were discovered in the clot. Here is a tube communicating with a small ovarian cyst, a tubo-ovarian cyst. Here, again, I show you a typical specimen of a dilated and matted tube; it is a tortuous S-shaped tube, with its convolutions adherent together and to the

ovary. Two of the convolutions are opened and did contain pus. There was a question in this case whether it was of tubercular origin; some thought it was tubercular, others believed it to be gonorrhœal. These tubes may be merely the result of chronic salpingitis very much thickened and containing pus—in other words, a pyosalpinx; or may contain serum—hydrosalpinx. A hydrosalpinx may be a primary serous exudation, or it may be secondary to pyosalpinx, where the pus has become sterilised by being long pent up, and has lost its original characters. Then the tube may be the seat of blood, a hæmatosalpinx, as occurs during the formation of a tubal mole. I have also opened *per vaginam* and drained tubes containing blood-stained serum, the origin of which was obscure, but which have closed up and given no further trouble. A tube may also rarely be the seat of a primary growth—an adenoma or papilloma, or even a primary carcinoma.

Amongst other movable lumps we may have masses of scybala; the small intestine coming down into the sacral hollow, or even the sigmoid flexure.

Then true hydatids have been described, apparently secondary to hepatic hydatids. I have seen one case in which the left kidney was sufficiently mobile to be felt in the sacral hollow. Needless to say it was not diagnosed, though it was suspected. We could not feel the left kidney, though that was no proof that it was not in its proper position, and it was thought possibly it might be that, though it was impossible to be sure until an exploratory opening was made.

A swelling of a malignant nature growing from the front of the rectum or from the small intestine, coming down until it became adherent to other organs, would be at first mobile. Pedunculated fibroids may be very mobile. Not very long ago I removed a fibroid which had no real attachment to anything. A patient had a lot of pain in the pelvis, and she had a movable tumour, a tumour which one could move about in every direction, and apparently it was not growing from anything. On opening the abdomen I found it was a calcareous fibroid about as big as a goose's egg, with a few adhesions, which were loosely connected with the bowel, but with absolutely no connection with any organ at all. It got its blood-supply from these few vascular adhesions amongst

the intestines. It had been shed from the wall of the uterus; one could see where it had been attached, and there were one or two other fibroid nodules on the uterus, which made it certain that that was its starting-place.

Now, how are we to examine such cases as I have been speaking about? The first step is to make an abdominal examination. You notice, perhaps, that respiratory movement in the abdomen is impaired, as it would be, for instance, in some inflammatory cases. Or you notice that the abdomen is distended, and possibly it is tender. You may notice that in acute inflammatory cases there is a distinct tumour in the hypogastric region, or over Poupart's ligament, and you will find on percussion that that tumour is resonant. On listening over it you will find it evidently contains gases moving about. You may also notice that that tumour varies from day to day, or even from hour to hour. You may notice it becomes much smaller after flatus has been passed, or after defæcation, than at other times. Such a swelling would certainly be matted bowel, a very common condition to find in cases of pelvic inflammation of all sorts, and in many other pelvic swellings (*e.g.* hæmatocele). It is a conservative arrangement by which the general peritoneal cavity is shut off; so that a woman may have a pelvic abscess of a very large size, or a pyosalpinx full of gonorrhœal pus, for instance, or an acute ovarian inflammation, or, perhaps, an ovarian abscess, and yet she has no general peritonitis. You may drain that woman's pelvis *per vaginam* without opening the abdomen at all, and it acts like an abscess anywhere else, for it is shut off from the general peritoneal cavity by the brim of the true pelvis being roofed over by matted bowel.

Then when you come to examine *per vaginam* with the finger, you will notice that matters are somewhat different from the normal. If it is a lump in the posterior part of the pelvis, which is what we are chiefly discussing, you will find that the uterus is pushed forwards and very likely upwards, so that the cervix is not easy to reach. Then you feel behind the cervix of this body a swelling, the nature of which we are supposed to be trying to interpret.

Now we have to find out a good many things about this particular lump behind the uterus

before we can decide what it is. For the purpose of ascertaining the size and shape of an organ, we try to approach it from many sides, to get a perspective view of it, as it were. For this purpose we use the bimanual method of examination, so as to get to know what the other side is like, and the relation between the tumour and each of your two hands, the distance between, and so on. Then you get another element in the perspective by examining *per rectum*. If you pass the finger rather high up in the rectum you can get to know the thickness of the structure from the vaginal to the rectal finger, or from the rectal finger to the abdominal hand. In this way you get to know the aspect of the tumour from three points of view.

We will, therefore, consider some of the points which we have to determine in making a bimanual examination of such a case. We will suppose that we are examining *per vaginam* and *per rectum* with one hand, either with one finger in the vagina and the other finger in the rectum at the same time, or with a finger alternately in the vagina and in the rectum, whilst the other hand is on the abdomen. You will thus have made an abdomino-vaginal, or an abdomino-rectal, or a vagino-rectal, or vaginal-recto-abdominal examination.

The first thing one wants to know is concerning the *position* of this lump. I need scarcely say that in a difficult case you have to examine under an anaesthetic, otherwise the abdominal parietes are so tense, especially if there be anything inflammatory, that the patient will not let you reach the parts.

The position of the tumour must be regarded in two ways: the position in the vertical plane, and the position in the horizontal plane. In the horizontal plane we want to know more particularly, is this tumour behind the broad ligament, or is it in it, or is it in front of it? You know where the broad ligaments come off from the uterus, exactly at the sides, and that the tendency of the broad ligament in health is to go backwards and outwards towards the sacro-iliac synchondrosis on each side. So the posterior quarters of the pelvis are comparatively small. We know, too, that the broad ligament is a lax structure, and that any tumour behind the broad ligament tends to push that broad ligament forwards, and there comes the

great difficulty of being able to be sure whether the structure is in the broad ligament or behind it. Some structures dip or burrow in the broad ligament; for instance, some fibroids growing from the sides of the uterus become intra-ligamentous. Most parovarian cysts burrow; a few paröphoric hilum cysts of the ovary burrow. In order to know whether a cyst is burrowing or not, one has to judge more particularly of its mobility. Any cyst which is burrowing in the broad ligament comes down and involves the connective tissue of the floor of the pelvis, and is embedded in it, and will not move about freely; and, moreover, it pushes the uterus over to the opposite side, which a body or cyst in the posterior quarter of the pelvis does not. You will at once see how that must be; that if a cyst is at all free to move behind the broad ligament, it will push that broad ligament forwards, it will push the uterus a little forwards, but it does not get fully to one side, so as to push the uterus body over to the other side. *Per rectum* we can pass the finger up and get behind any cyst, whether it is in the broad ligament or just behind it, and you are sometimes able to bring the rectum away to one side so much that you can get on the outer aspect of the cyst, and are then able to judge exactly as to its relationship.

Then we have to judge as regards the vertical plane. Our object more particularly is to determine whether it is sub-peritoneal, lying beneath the peritoneum in the cellular tissue or in the pelvic peritoneal cavity. A bi-manual examination will almost always determine the point, because the tumour is so much more definitely fixed to the floor of the pelvis in cases where it is free in the peritoneal cavity.

If it is a fixed tumour we have to judge rather by the level at which it is, because it may be fixed by perimetritic inflammation. In such a case rectal examination, by which you are able to get well above the level where the peritoneum is attached to the pelvic floor, enables you to feel whether it is above or below it.

Secondly, to determine the *mobility* of a pelvic tumour, a bimanual examination is necessary. We are able to grasp the swelling between the two hands, especially if the patient is under an anæsthetic, and are able to lift it away as it were, or to depress it irrespective of other surrounding organs. If you have two fingers in the vagina, for instance,

one finger being applied to the cervix uteri and the other behind on the tumour, you may be able to feel the fundus of the uterus anteriorly, and you may be able to move the uterus quite independently of the tumour; and you may then transfer your attention to the other finger, which is on the tumour, and then you are able bimanually to lift that tumour up and down, and perhaps move it to one side or bring it away from the uterus. You may find when you try to move the tumour that the uterus moves with it, and that it is only to that extent fixed. Or you may find it so fixed to everything that when you move the tumour you lift up the vaginal roof or pelvic floor, and perhaps drag on the rectum. By such means you can ascertain not only its actual but its relative mobility. You may find it is apparently fixed, but when you come to elevate it that it is only impacted, as in a retroverted gravid uterus, and when you place the patient under an anæsthetic in the Sims's position, and allow gravity to act forward towards the abdomen, and allow atmospheric pressure to act by letting air enter the vagina, you will find you are able to push that tumour up, and what was apparently fixed becomes mobile, because it was simply impacted.

The third thing we have to find out is the relation of the tumour to the central pelvic organ, the uterus. That can, as already stated, generally be ascertained bimanually. Sometimes it is not so easy as it seems, and one may have to draw the uterus down with forceps, and with one's finger on the tumour *per rectum*; you will find such traction does not interfere with the tumour behind, or that it only interferes with it after the uterus has been drawn down for a certain distance. For instance, it may be a prolapsed ovary; or it may be a pedunculated fibroid. You find when you draw the uterus down that it does not interfere with the fibroid until you have gone a certain way down, and then you find any further movement of the uterus moves the fibroid, and you can tell it is something with a pedicle growing from the uterus.

Another good way is to pass a sound into the uterus under antiseptic precautions, and you may find that when you move the sound the swelling moves also. The swelling is part of the uterus, such as a fibroid. It might be a prolapsed ovary. Then you may find when you draw the uterus over in one direction that it does not move the ovary at

all. Moved in another direction it will drag that ovary, and you can tell to which corner of the uterus the tumour is most attached.

You are often able to tell by some one or other of these methods the exact relation of the tumour to the uterus. If the whole thing is fixed you will do very little good by trying to find out these relations, except that if you pass the sound and it goes forward, and there is still this mass behind, you know it is not a fundus uteri fixed by inflammation, and that it must be something outside the actual uterus itself, or an impacted fibroid in the posterior wall of the uterus.

The *size* and *shape* of the tumour is made out by one or other of these bimanual methods, just taking a perspective of the tumour from different points of view. Fibroids are generally irregular in shape, ovaries are pretty uniform. As a rule ovaries are ovoid in shape, supposing you can map them out sufficiently to determine that. Fallopian tubes when they are swollen, may be, in a typical case, as in pyosalpinx, banana-shaped, but are by no means necessarily so. If you look at this case of matted tube, which is not at all an unusual condition, you will see that you could not tell that it was a tube by any method of examination; you would only know there was a tumour of ovoid shape, which might or might not contain a tube.

Next with regard to *consistency*. One has to try to ascertain whether it is fluctuating or merely elastic, or whether it is very hard. If it is very hard it is either an inflammatory exudation or hæmatocele or an unusually hard fibroid. If it is fluctuating it may be a serous exudation, it may be pus, or, rarely, it may be recent blood. Such a fluid may be either in the Fallopian tube or in Douglas's pouch, or in an ovarian abscess, or if superficial possibly in a large vaginal cyst.

Pain on examination must also be considered. Pain is a very unreliable symptom in the female pelvis. The pain is often referred to exactly the opposite side of the pelvis to that diseased, and the same is well known to occur in kidney cases. On more than one occasion a right kidney has been cut down upon expecting to find a calculus there, because of pain being complained of in the right side, and yet, eventually, the stone has been found in the left kidney, the result, possibly, of a poorly discriminating sympathetic system. Women generally complain of pain on the left side of the

pelvis, whichever ovary happens to be prolapsed. Why they should complain of pain more on one side than on the other is not very clear. In prolapsed ovary the pain is very characteristic; contact gives a sickening sensation very much like that which we know as a testicular pain. The patient will also tell you the moment any acute inflammatory exudation is touched during examination, but you have to decide whether it is due to perimetritis, to ovaritis, or to salpingitis, etc.

Amongst the common swellings, the only ones we will speak of to-day are the Fallopian tube swellings. I have had a good many of these in Chandos Ward during the last year or two here. They generally cause more or less fixation of the uterus, because there is a certain amount of associated perimetritis, and I think their chief characteristic is their recurrence. A patient gets an attack of gonorrhœa; she has some vaginal discharge, perhaps urethritis, too; the uterus becomes tender and inflamed, so that sitting is painful, coitus is painful, and she becomes more or less of an invalid, but without much pyrexia or anything very much to show for it, either objectively or subjectively. Then, perhaps just before or just after a period, she gets a rigor, with acute abdominal pain, and we find some matted bowel over the lower abdomen and, in a day or two, *per vaginam*, a fixed uterus with retro-uterine induration. She may or may not go through an extremely severe attack of acute perimetritis, but the majority of the cases clear up after five or six weeks, and she gets to some extent better. Then after a month or so she gets another attack, each attack usually not quite so severe as the last, because the inflammation is limited in area by pre-existing adhesions. Any one of these recurrences may give rise to an intra-peritoneal pelvic abscess, which may open into the vagina or into the rectum, or into one of these coils of small intestine. What has happened? The inflammation has spread from the uterus along a tube, and she has got a more or less acute salpingitis. The inflammation spreads to the fimbriated end of the tube, and that causes the peritoneum which covers the edges of the Fallopian tube to become involved. Then either a definite abscess forms there, or, what is much more common, some adhesive lymph is thrown out, which covers in the end of the Fallopian tube, or, if you like, fixes it to some surrounding organ, such as the

ovary, and then one gets the fimbriated end of the tube everywhere adherent, or the end of the tube rounded off with the fimbriæ inside. In either case, the end of the tube becomes completely obliterated. Here is a very good case of obliterated end of a tube with a rounded end and its fimbriæ effaced, with the resulting formation of a pyosalpinx. Here is a specimen of a recurring chronic salpingitis, and the fimbriated end is again obliterated, with much lymph floating about evidencing perimetritic exudation. Sealing up of the end of the tube, often without further extension of inflammation to the peritoneum, generally results in a pyosalpinx, that is to say, pus collected in the tube. Occasionally this pyosalpinx (or hydrosalpinx) opens again into the uterus, and you get a recurring discharge through that into the vagina. Some deny that that is so, but I have no doubt at all that this does occur. And it is easy to explain that it may occur, because in most cases of even large pyosalpinx you can pass a bristle from the uterus into the tube. The tube at the uterine end does not seem to be closed, but it is closed by a valvular method, that is to say, by some of the redundant mucous membrane of the tube fitting into the little opening.

The treatment of pyosalpinx has undergone a great deal of change in modern times. In the olden days of abdominal operations attempts were made to remove all these tubes, and they were the most dangerous operations in pelvic surgery. If these tubes were removed in the acute or sub-acute stage, before the pus became sterile, and the tubes burst during the operation, the gonorrhœal pus was discharged all over the bowels into the peritoneal cavity, and the patients almost invariably died; at any rate the mortality of the operation was 25 per cent. to 30 per cent. It is a very difficult operation to disembel these adherent tubes, and the risk of rupture is great. At this hospital I have dealt with a considerable number of these tubes by vaginal drainage, and if you find a tense, fixed swelling in the retro-urine spaces of the pelvis in close contact to the posterior wall of the vagina, it is far better and safer to explore by posterior colpotomy (or, if preferred, by anterior colpotomy), opening the vagina behind the uterus; get your finger on to the cyst itself or collection of fluid, and look at it if you like by means of a speculum, and then,

either remove it, if not very adherent, or merely incise it and drain. There are many cases now not only of complete recovery—because as far as I know they always recover—but of functional activity of the tube, and even of pregnancy taking place afterwards. I remember one case particularly of a lady whose husband had a chronic gleet. She had several attacks of perimetritis; and on one occasion the tubes on both sides became definitely swollen and inflamed, and finally, as far as one could tell, they each developed into a pyosalpinx. I opened the vagina posteriorly, and as far as one was able to judge—both myself and my assistant came to the same conclusion—it was a double pyosalpinx. I incised both tubes, and put a drainage-tube into each of them, treating them as ordinary abscesses. After a time the drainage-tubes were removed, and the case did very well. A year or so afterwards she became pregnant and miscarried, owing to the uterus being fixed by adhesions. In that case, supposing one was right in the diagnosis, these tubes had so far recovered their functions that pregnancy had occurred. There are several other cases recorded in which not only has the patient recovered from the constantly recurring condition, but all her functions have also resumed their normal state. So it is not nowadays required to disembel a pyosalpinx when it is definitely fixed low in the pelvis. Sometimes, though rarely, they are very large, involving the lower part of the abdomen, and are not really in the pelvis at all. I am not speaking of those to-day. Provided the retro-uterine lumps are in the pelvis, so that they can be reached *per vaginam*, that is the way they should be approached. We had a case the other day which probably was a case of this sort, or it may have been a parovarian abscess. The patient had been in the hospital off and on, under my observation since 1884, with recurrent attacks of perimetritis. She had a cystic body of some sort in the pelvis, which was very tender, and her life for the past sixteen or seventeen years had been a burden to her. But I have never had her in the hospital until a fortnight ago; and then I found that she had an attack of acute perimetritis *plus* this collection of tense encysted fluid. One day when I came she had a very definite fluid exudation in the pelvis outside this cyst or tube; so I opened the

posterior pouch of the vagina and let out several ounces of fluid, and then I found this cyst, which apparently was covered by either inflamed peritoneum or a thin layer of broad ligament, I was not sure which. At all events, I incised it freely, and three or four ounces of pus came away. That may have been an old pyosalpinx, but it was so matted and adherent to everything that one could not find out its exact relations. I have drained it with a drainage-tube, and now she has got a diverging stem pessary in the sinus, because the drainage-tube has come out, and I have no doubt she will make a perfectly good recovery, and I hope without any of the illness and invalidism which she has had. I am convinced that this is the way in which these cases should be approached under the conditions named.

Spinal Analgesia.—William Seaman Bainbridge reports twelve operations on infants and young children during spinal analgesia. They were performed for the relief of phimosis, inguinal hernia, prolapse of the rectum, genu varum, talipes equino-varus, tuberculous arthritis of knee-joint, with contracture of the flexor tendons of the thigh; cystitis and vesical calculus, and cellulitis of left calf, with a discharging sinus leading to dead bone. The author finds that children of all ages stand this method exceedingly well. They may cry out before or during the injection, but they soon quiet down, and usually remain calm during the operation. In all cases the ether method of sterilisation of cocaine and eucaïne was employed. A short, bevelled needle having a steel point, the remainder of the needle down to the shank constructed of a soft metal, was used, in order to avoid the possible danger of breaking should the patient struggle. The author has given what seemed at the time large doses of cocaine, but in only one instance did any alarming symptoms occur. In this case it was impossible to say whether the injection was the cause, or the disease from which the patient was suffering. Usually the increase in the drug has resulted in a more satisfactory analgesia and fewer after-effects. One of the cases reported is the second instance on record of general analgesia. The writer has found beta-eucaïne to be less reliable than cocaine.—*American Journ. of Obstet.*, October, 1901.

HEART SYMPTOMS IN NEUROTIC SUBJECTS.

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CASES of heart trouble in neurotic subjects demand more than ordinary care in their investigation, since success in treatment depends entirely on correctness in diagnosis. Their recognition and management require observation and tact no less than careful physical examination. Nor is the prognosis less important, for fears raised, or rather increased, by an unjustifiably bad prognosis will militate strongly against the patient's recovery, while an assurance of a return to health on insufficient grounds would be disastrous to the patient and damaging to the practitioner's reputation. These patients often protest they are quite prepared to hear the worst although it is abundantly evident that such is not the case. It may be difficult in some cases to arrive at a satisfactory diagnosis, especially on the first examination, and it is better to postpone giving an opinion than to send the patient away with the diagnosis "a weak heart," because the vagueness of such a diagnosis, instead of being a comfort to the nervous patient, is a constant source of anxiety to him.

CASE 1.—A science teacher, æt. 25, complained of palpitation coming on at odd times, without—as far as he knew—any definite cause. There was occasionally shortness of breath on going upstairs. He was a well-developed man of a neurotic temperament. He had always been great at sports, but under medical advice he had avoided them for over a year. He was very anxious about his health, and was desirous above all things to resume cricket that season if possible. He had been a heavy smoker, consuming generally four ounces of tobacco a week. He had never had a severe illness, nor any feverish attack. Two years before, at the end of the rowing season and after a heavy day with the sculls, he had had an attack of palpitation, and the heart did not settle down for several days. The anxiety caused by this symptom was much increased by having been told that his heart was enlarged. In the course of his biological studies the patient had

recently dissected a heart, so it would have been singularly inopportune to have given him an alarming opinion of his case at that time. A month's rest and treatment sufficed to remove the unpleasant symptoms and to restore him to his usual health. He remained well until he had an attack of influenza, after which he had a fainting attack at the end of a long day's reading. For a year he had taken no violent exercise. His lungs and abdominal viscera were normal. The heart's action was full; the apex was under the sixth rib a little internal to the nipple line; the cardiac dulness extended to 2 cm. to the left of the same line; the dulness was normal on the right side. Pulse 78, regular, and of good volume and tension. Auscultation, before and after the patient had been submitted to exercise, gave a negative result. The liver was not enlarged. The urine was normal. *Diagnosis.*—Hypertrophy of the left ventricle, little or no dilatation. The hypertrophy was probably due entirely to the demands made upon the heart during a long course of athletics. The palpitation was due to indigestion, and the faintness to excessive smoking, the toxic effects of the tobacco being probably less easily resisted since the influenza. The *prognosis* was favourable. *Treatment* was threefold—mental, dietetic, and medicinal. The most important thing was to impress on the patient's mind the true significance of the cardiac hypertrophy; secondly, simple directions as to diet were given, smoking was limited to two pipes daily, and those after meals. The medicinal treatment was a mixture containing arsenic, bromide of ammonium, and iron, to be taken thrice daily. As regarded exercise he was to play cricket moderately, and if, after a week or two he found no inconvenience from playing, he might return to full enjoyment of the game. The patient was not able to come again until nearly three months after the first consultation. He was very much improved in health and spirits; he had played a match three weeks after commencing treatment, and he had played twice a week ever since. On no occasion had he felt any inconvenience from his heart, in fact he at times forgot he had one. He could walk ten miles with ease. If he passed much beyond his regular meal-time there was a little uneasiness at the lower part of the cardiac region, which was speedily relieved by taking food.

This case was undoubtedly a very mixed one; a neurotic temperament, the heart often subjected to severe and prolonged strain, indigestion, and tobacco poisoning. There was also a possibility of there being some post-influenzal effect on the circulatory system. It would be difficult to say which of these factors was the most responsible for the symptoms presented by this patient.

As neurotics are generally very active people, this patient was an enthusiast at cricket; and, as another characteristic of the same temperament, he was easily depressed. The heart was taxed by exertion and its action inhibited by nicotine. Small wonder that the cardiac action was at times irregular and at others boisterous. The moderate hypertrophy of the left ventricle was, in the absence of valvular, arterial, or renal disease, physiological.

The chief points to be considered in a case where increased cardiac dulness exists are:

1. The patient's physical development.
2. The history, especially as to peri- or endocarditis.
3. The state of the valves.
4. The position, character, and strength of the apex-beat.
5. The relative amount of dilatation.
6. The part or parts affected.
7. The condition of the other viscera.
8. The condition of the pulse as indicating
 - (a) the tone of the heart muscle, and
 - (b) the state of the arteries.
9. The condition of the urine.

The enumeration of the above points is sufficient to expose the fallacy of much that has been impressed on the minds of patients in some quarters as to the necessarily grave import of the discovery of cardiac enlargement, and that the great object of treatment—nay, the prolongation of life beyond a brief period—depends upon a demonstrable lessening of the area of the cardiac dulness. If, during a course of special baths, etc., a patient has any doubts as to the beneficial effects of the treatment, an appeal is made to charts and sphygmograms into which the personal factor has probably largely entered. My patient would have been prostrated if his cardiac hypertrophy had been made much of, and he would have spent his last penny in seeking to obtain the coveted reduction if he had been impressed with the vital necessity

of so doing. For a diagnosis to be of any real value as a guide to prognosis and treatment, percussion, auscultation, and pulse tracing must not solely be depended upon; neither are the results of treatment to be estimated by the same imperfect means.

CASE 2.—A retired publican complained of nervous depression, giddiness, stopping of the heart, weight at the epigastrium, with flatulence. Whiskey generally relieved him. Beyond nervousness and constipation he had never been ill. The patient was agitated on entering the consulting room. He had not the appearance of a confirmed alcoholic. He said he had lost heavily on some speculative investments. The tongue was furred, bowels costive, appetite bad, and he slept badly. Pulse 80, tension not markedly increased; urine acid and devoid of albumin. Nothing abnormal in chest or abdomen. *Prognosis*, good. *Treatment*—(1) Positive assurance that there was no organic disease of the heart. (2) Careful dieting with abstention from alcohol excepting a small quantity with meals. (3) An aperient every night and a stomachic mixture with bromide three times a day before meals. Under this treatment the patient much improved and had better nights, and by persevering with the treatment the improvement continued, and in six weeks' time he was as well as he had been for several years prior to his illness.

The patient inherited a nervous temperament; he had accumulated money, retired from business, and subsequently lost a large part of his savings, and being out of business he had nothing to occupy his mind, consequently he was continually brooding over his financial and bodily troubles. The measures adopted were directed towards the removal of the patient's morbid apprehensions, calming of the nervous excitement, relief of constipation, and improvement of the digestion.

CASE 3.—A thin, spare man, æt. 56, complained of loss of strength and sudden attacks of trembling, with uneasiness at the region of the heart; his breath was short at times, but not specially so on going upstairs, there was giddiness on rising in the morning, and a sinking sensation at the epigastrium. The symptoms were more in evidence two hours after meals. He had had a phosphatic calculus removed from the bladder three and a half years before, but there had not been any other illness. He had taken tea to

excess for many years. He frequently tested his urine to see if it were alkaline. He was very excitable, and took a serious view of his case. His teeth were deficient; his bowels were regular, and his lungs healthy; the heart's action was feeble and intermittent; the apex was in the normal position; there was no increase of cardiac dulness; the first sound at the apex was a little prolonged, but there was no murmur; the liver was not enlarged; the urine was loaded with phosphates, but there was no albumin. The *prognosis* was good. *Treatment*.—To cease feeling his pulse and examining the urine, and generally to think less of his ailments; to take plenty of exercise in the open air; to drink cocoa instead of tea; and to have a liberal supply of meat. Strychnine with mineral acid was ordered to be taken three times a day, twenty minutes after meals. Under this treatment (the medicine having been varied from time to time) the patient's health greatly improved, and the urine ceased to be alkaline.

Here was a man of thoroughly neurotic type, and of the so-called phosphatic diathesis, who was over-solicitous about his health, noting symptoms and attributing grave significance to them all, especially those connected with the heart. He was an excessive tea drinker. It is to be noted that exertion neither provoked dyspnoea nor increased it when present as in true cardiac cases. Phosphatic urine is not uncommon in neurotic patients, and in cases of long-standing dyspepsia, the free use of alkalies, or an almost exclusive vegetable diet are possible causes of that condition.

CASE 4.—A farmer, æt. 45, complained of a sense of discomfort at the region of the heart, palpitation on lying down in bed, and a difficulty in getting to sleep, a sinking sensation at the epigastrium, and mental depression. These symptoms were ushered in seven weeks previously with faintness and giddiness without any assignable cause. There was no history of former illness beyond influenza four years before. The family history was that the mother was highly nervous. He had drunk heavily of spirits for eight years, and his allowance was four glasses of whiskey daily. He was a well-nourished man with a florid complexion, who took his meals regularly, and stated that his bowels were regular. The chest was normal; the liver was a little down, and there was tenderness on pressure at the epigastrium; the urine was

normal. The molar teeth were nearly all gone. *Treatment.*—To have artificial teeth; to take his principal meal in the middle of the day and no food after 7.30; to take no stimulant beyond a glass of claret with his meals. A dose of calomel was ordered to be taken once a week, and an alkaline stomachic draught three times a day before meals, and a pill of valerinate of zinc night and morning. He was much improved a week later, he had only been giddy once. The calomel brought away a large amount of scybalæ. An equally satisfactory account was given three weeks later. The patient was not seen again until eight months later, when he was found to have signs of melancholia. He was ordered to take a voyage accompanied by a judicious friend. Unfortunately I have not heard of or from him since.

There is generally much difficulty in obtaining the mental side of a family history. In this case I am inclined (in the light of its after development) to the view that the "extreme nervousness" in the mother was a form of insanity. The amelioration under treatment was very striking, especially when the teeth were supplied and good mastication obtained. And here I would urge the importance of examining the state of the mouth in all cases, for the presence of bad teeth, or the want of good ones will often explain the cause of obscure pains and intractable indigestion. Finally, bodily and mental improvement always follows the removal of scybalæ by a calomel purge.

Operation for Cystocele.—In the operation used by Emerson M. Sutton the incision is made around the cervix, extending to the lateral sulci, forming the base of a triangle, the apex of which is at the meatus, the sides of which take in sufficient of the anterior wall, when the edges reunited will completely retain the superimposed bladder; denudation made as one flap; amputation of the cervix, or repair of the cervix, as the case may require, being performed at the time of the first incision, and dissection of the tissues from the anterior surface of the cervix sufficiently to allow the easy reposition of the uterus. The result of this operation is a lengthening of the anterior wall of about three centimètres. This operation places the uterus in its natural position, and does away with the need of suspension or shortening of the round ligaments.—*Amer. Gyn.*, August, 1901.

ON THE CAUSATION, SYMPTOMS, AND TREATMENT OF ACUTE AND SUBACUTE BRONCHITIS.

By J. COURTENAY MACWATTERS,
M.R.C.S., L.R.C.P.

BRONCHITIS is a catarrhal inflammation of the mucous membrane and subjacent tissues of the bronchial tubes, involving those of greater or smaller calibre, and is usually bilateral.

To thoroughly understand the pathological changes and symptoms occurring in bronchitis, it will be necessary to consider the anatomical relations of the tissues involved.

That the inflammation shows no marked tendency to invade the pulmonary tissues surrounding the tubes, is explained by the fact that the lungs have a double vascular supply, the bronchi being supplied by branches of the bronchial arteries, from the thoracic aorta, while the air-vesicles are supplied by the capillary plexus of the pulmonary vessel. Moreover, the bronchial arteries participate in the general vaso-motor control of the systemic vessels, while the pulmonary artery is not subject to such variations; consequently, a variety of causes, acting either directly through the bronchi, or indirectly by various vaso-motor reflex actions, are able to cause a vascular engorgement limited to their distribution, viz. the bronchial tubes.

The bronchi divide dichotomously into fine ramifications, the minute bronchioles ending in infundibula and air-vesicles. A section of a medium-sized bronchiole shows three coats—an inner *mucosa*, a middle *muscularis*, and an outer *fibrous*, containing the annular cartilages. Further examination shows that the mucosa is composed of (1) an epithelial lining with three layers of cells: (a) Internally, ciliated and mucous; next, (b) transitional or rounded cells actively germinating to form ciliated or mucous cells; and (c) a single deeper layer of nucleated cells lying on (2) a fibrous layer, consisting of numerous bundles of fibrous tissue with intermediary lymphatic spaces containing many lymphatic corpuscles. The mucosa is effectively separated from the vessels and lymphatics of this inner fibrous layer by a structureless basement membrane, giving attachment to the epithelial covering. According to J. D. Hamilton ('Pathology of Bronchitis,' 1879),

it is this basement membrane that imparts a superficial character to the catarrhal affections of the bronchi; it also prevents the passage of inhaled particles, like coal dust in coal miners' lung, being carried through to the tissues of the bronchi. Such particles can only enter the lung through the air-vessels.

The distribution and actions of the muscular layer is of certain aid to us clinically. This layer is most marked in the small bronchi and bronchioles, and is continued down to the infundibula, where it is evidenced only by a few scattered fibres. The physiological action of these muscles is not definitely known, but it is probably of importance in regulating the intra-alveolar air tension and in protecting the delicate air-vesicles from undue or sudden pressure in coughing, or other forced expiratory acts; and this is borne out pathologically by the facts that this coat is much hypertrophied in chronic bronchitis, and that in fatal cases of bronchitis, where the muscular coat has lost its functional activity, the bronchial tubes dilate and become filled with secretion.

D. Walsh (in his 'Diseases of the Lungs') refers to Radcliffe Hall's researches proving that in health the muscular coat is in forced expiration capable of reducing the calibre of the tubes below that of their quiescent state, and points out that the act of expectoration would be greatly impaired should this muscular layer lose its contractile force and the elasticity of the bronchi become diminished. This, then, explains the dilatation of the bronchial tubes, the difficulty of expectoration and consequent accumulation of mucus, and the laboured expiration of any severe case of bronchitis.

Watson Williams thinks that there is probably a rhythmic action of these bronchial muscles allied to that of the *alæ nasi*, dilating and contracting the tubes with inspiration and expiration, and he believes that "this largely explains why, in bronchitis and asthma, the dyspnoea is expiratory rather than inspiratory, inasmuch as the spasm is more or less inhibited during inspiration, permitting air to be inspired more freely than it can be expired during the period of bronchial spasm, consequently the lungs become distended and emphysematous."

On following up a bronchiole through its finest ramifications to the air-vesicles, we find that the

muscular coat becomes relatively scanty and much thinned out; the columnar ciliated epithelium gives place to cubical in the alveoli, and finally squamous epithelium in the vesicles, where the muscular coat is entirely lost. This thinness of the walls of the smallest bronchi, their small calibre, the absence of ciliated epithelium and of muscular fibres, explains the danger arising from a bronchitis involving the capillary tubes. The basement membrane giving attachment to the epithelial covering also gradually disappears, being lost in the alveoli and vesicles, so that it is impossible for a bronchitis of the capillary tubes to be present without involvement of the surrounding pulmonary tissues, in which pneumonic areas appear, giving rise to a true broncho-pneumonia, and the term "Capillary Bronchitis" of the older textbooks will be, therefore, seen to be both misleading and erroneous.

Ætiology.—The causes of bronchitis may be divided into: (A) *Predisposing*; which again may be (a) *atmospheric*, sudden change, as from hot to cold weather—cold, damp, and foggy weather being injurious,—east winds, life in a vitiated atmosphere, whether the impairment be due to animate particles, as micro-organisms, or inanimate, as dust, etc.; and (b) *individual* causes, as the extremes of age—the very young and the very old being specially liable to this complaint,—indoor life, sedentary habits, extreme care in wrapping up, commonly termed "coddling," which gives rise to increased sensibility to cold; heart disease, especially those forms which cause passive congestion of the lungs; gout, rheumatism, alcoholism, syphilis, diabetes, "catarrhal diathesis," the causes of mouth breathing, such as adenoids, enlarged tonsils, polypi of the nose, etc.; the period of dentition, and above all, previous existence of pulmonary affections and previous attacks of bronchitis. (B) *Exciting*, exposure to cold, inhalation of steam or irritating particles or gases, toxæmia of gout, rheumatism, Bright's disease, typhoid fever, septicæmia, etc.; and, lastly, infective, where the condition is due to the direct action of the micro-organism, as in influenza, enteric, typhus, measles, scarlet fever, small-pox, malaria, whooping-cough, and diphtheria.

By far the greater number of cases of bronchitis may be traced to exposure to cold; but this, alone, is certainly not to blame for the ma-

majority of cases attributed to "catching cold," for we find that it is by no means those persons who are daily exposed to great and rapid changes of temperature that are most prone to bronchitis; in fact, persons who live an out-door life and who do not "wrap up" are, as a rule, less liable to this malady than those who live an in-door sedentary life. Cases of acute inflammatory catarrh of the upper respiratory tract are generally found in persons who have been attending an overcrowded and ill-ventilated place, and are then exposed to cold; "colds" being very rarely found in persons living in a pure atmosphere, as on the upper Alps, or at sea. It has been shown that the micro-organisms present in normal sputum, mouth, and air-passages, viz. streptococci, staphylococci, leptothrix, spirilli, chromogenic bacteria, bacilli and sarcinæ, greatly increase in number and virulence in damp weather and winter months, and are then capable of setting up acute pharyngitis if applied to the throat.

This, then, explains the frequency of attacks of bronchitis in the changeable weather of early spring and late autumn. In the sputum of bronchitis in early and mid-stages by far the most prevalent micro-organisms found are the streptococcus, and Fränkel's pneumococcus has been found in 15.85 per cent. of cases of so-called "capillary bronchitis."

The most reasonable theory (Netter) seems that bronchitis is due to the invasion by streptococci and staphylococci of a mucous membrane whose resisting-power is reduced, owing to reflex congestion set up by a variety of causes, and I am convinced that in by far the greater number of cases this is started by exposure to cold.

The bronchitis following the inhalation of dust in unclean, ill-ventilated rooms is probably due to the fact that the dust contains numerous morbid organisms in addition to the inanimate particles which set up an irritation of the air-passages, and that these microbes, then attacking the injured mucous membrane, become active and virulent, thus setting up the catarrhal inflammation, which then spreads down the trachea and bronchi.

Doubtless a few are due in some degree to the actual inspiration of cold air, as evidenced by the experience of some susceptible persons going from a heated atmosphere into cold night air, the

frequency of attacks in habitual "mouth-breathers," owing to the inspired air not being duly warmed by its passage through the nose, and as evidenced by the susceptibility of tracheotomised patients; but in by far the greater number of cases the action of exposure to cold seems to be indirect rather than from the direct inspiration of cold air, and is due to vaso-motor paralysis, as pointed out by Rosenthal, and evidenced by cases due to getting wet, lying on wet grass, sitting still in a draught, especially if perspiring; and this is only analagous to the liability to inflammatory catarrhal attacks of other organs—liver, bladder, intestines, etc.—from similar reflex vaso-motor paresis due to exposure to cold.

As regards the secondary bronchitis arising during the course of the specific fevers, these are probably set up by the toxic products in the circulation.

Morbid Anatomy.—The initial stage is universally agreed to be hyperæmia with concomitant dryness and swelling of the mucous membrane, followed by excessive excretion, and it is in the branches of the bronchial artery in the attendant plexus of blood-vessels ramifying in the inner fibrous coat just beneath the basement membrane that this relaxation and distension takes place. Socoleff and Hamilton found that quite early in the course of the disease the ciliated epithelium desquamates, undergoing fatty degeneration, and is then for the greater part expectorated, some remaining loose to be inhaled into the smaller bronchi to form plugs with other catarrhal products. Wilson Fox and others consider this desquamation to be only a post-mortem change, and Hamilton admits that it is only a partial one, the shedding being seldom, if ever, complete; but as ciliated epithelial cells have been observed in the sputa of early cases of bronchitis, I cannot but think this desquamation takes place in the course of the disease quite apart from any post-mortem changes.

While these cells are undergoing proliferation and desquamation the mucous cells are particularly active, and it is this catarrhal mucus, combined with epithelial cells, which gives rise to the mucopurulent fluid which forms the sputum.

Should the condition of primary congestion and consequent catarrh fail to subside or yield to treatment, it was observed by Hamilton that by the tenth day the congestion not only affects the

inner fibrous coat, but all parts of the bronchus exhibit vessels over-distended and engorged with blood; the inner coat becomes infiltrated with the cells derived from the endothelium lining the lymphatic spaces until the whole lymphatic spaces become choked with these cellular products. The cells, according to Hamilton, do not extend to the free surface of the mucous membrane, owing to the impassable barrier afforded by the basement membrane, but extend outwards through the lymphatic spaces to the adventitia. This proliferation with the production of leucocytes soon spreads throughout the interlobular septa to the deeper layers of the pleura, the whole lymphatic vessels becoming involved, and the lymphatic glands at the root of the lung are then invariably enlarged.

In those cases which from the severity of the attack, or from the failure of those factors which militate for recovery; or in those less acute cases which, owing to their pertinacity, pass into the condition of chronic bronchitis, the mucous membrane and the entire thickness of the bronchial wall remain thickened, the muscular coat hypertrophied, and the tubes dilated, while the mucous glands more or less completely disappear, being replaced by dense cellular infiltration.

Symptoms.—Bronchitis may present us with every aspect, from a slight and transient "cold on the chest," to a severe and rapidly fatal disease, accompanied by rapid heart failure and collapse. In the milder cases the attack is usually ushered in by the symptoms of an ordinary "cold in the head," with "pains all over," frequent sneezing and lachrymation, and a soreness of the nasopharynx rapidly spreading to the pharynx as the catarrh extends downwards towards the larynx, with consequent hoarseness and loss of voice, and to the trachea and bronchi, giving rise to a sense of tightness and rawness behind the sternum, with a feeling of oppression about the chest. The early stages are sometimes accompanied by considerable gastric catarrh, hepatic congestion, and constipation; the pulse and respiration are moderately quickened, and the skin, at first dry and harsh with feelings of chilliness and frequent shivering, soon becomes moist. Occasionally the attack seems to begin "on the chest" without obvious symptoms of catarrh of the upper respiratory tract. More rarely, particu-

larly in the very young and very old, an attack may be quite sudden in onset, with severe dyspnoea and cough, almost resembling an attack of spasmodic asthma, except in its pertinacity; or again the cough and dyspnoea may set in suddenly without the marked spasmodic character.

The temperature, as a rule, even in moderately severe cases, is but little raised, but occasionally in the more severe and sudden cases it may reach 102° or 103° , with morning fall and evening rise.

The pulse is increased in frequency in the early stages, but rarely, except in severe cases, exceeds 110 or 120 per minute. In old and feeble persons with a damaged heart the pulse soon shows signs of grave disturbance, irregularity and intermittency commonly occurring.

The respiration is increased in frequency according to the extent of the tissues involved and the amount of spasm present, in the milder cases being about 24 to 28 per minute, while in infants it may in grave cases reach 40, 50, or even 60 per minute, usually with a pulse of 120 to 150. In infants and very young children there may be often noticed a peculiar catch at the end of inspiration, which is usually an accompaniment of broncho-pneumonia, and probably due to the involvement of the pleura.

The urine is high coloured, loaded with lithates and urea, of high specific gravity, and free from albumen.

The cough is the symptom of which the patient mostly complains. At first it is harsh and dry, either coming in frequent single coughs, or at longer intervals in severe repeated paroxysms, each lasting half a minute or more, extremely harassing to the patient, who is frequently quite exhausted after such attacks; attacks occasionally end in vomiting. It is in the earliest stages before expectoration begins that this cough is often most troublesome, at times giving rise to actual pain behind the sternum and at the lower edge of the thorax, at the attachment of the abdominal muscles and diaphragm. The cough is due to irritation of the dry inflamed bronchial mucous membrane, this irritation being referred to the laryngeal region.

Physical examination at this stage may reveal nothing, or perhaps a little sibilus or rhonchus may be heard.

After an interval, varying according to how its course is modified by treatment, the mucous membrane begins to secrete a little watery and frothy

serous exudation, containing a few ciliated epithelial-cells which are shed very early. Soon catarrhal mucus appears more abundantly, and the sputum quickly becomes "muco-purulent," so called, owing to the number of round-cells, epithelial cells being thrown off from the deeper layer. With the onset of expectoration the patient feels better, the increased rate of breathing and the dyspnœa disappear, the cough is less persistent and harassing, being now due, not to the irritation of the dry mucous membrane, but to the presence of accumulating secretions, which call for removal; hence the fact that it is more severe after recumbency and sleep. Streaks of blood are not uncommon in the sputum, especially in the early stage, when there is much cough.

While so-called "capillary bronchitis" may be remarkably sudden in its onset, it frequently commences with an ordinary mild bronchitis spreading to the smaller tubes. This tendency has been specially noticed in the bronchitis occurring in some epidemics of influenza (Burney Yeo). In severe cases nervous symptoms arise early, and are pronounced. Restlessness, muttering delirium, and sleeplessness at night are common, while drowsiness is not infrequent during the day; maniacal excitement may occur; complete loss of appetite is common, and the patient may rapidly become cyanosed and asphyxiated unless soon relieved.

The right side of the heart dilates if the attack be prolonged, or a large tract of lung involved, as evidenced by epigastric pulsation, or it may be pushed down, the apex being felt in the left costal angle.

The liver is often congested and pushed downwards, and an icteric tinge about the patient's sclerotics and face is often noticeable.

In the acute asphyxiating type the respiratory movements of the over-distended chest are increasingly shallow, and the heart's action is apt to fail rapidly, with marked cyanosis, clammy perspirations, and coldness of the skin.

Inspection and percussion as a rule are not modified in bronchitis; on palpation bronchial fremitus can often be felt.

There are two kinds of adventitious sounds that arise in bronchitis, dry and moist, rhonchus and sibilus being the dry, râles the moist. Rhonchus is the term applied to the loud, deep cooing sound, due to air passing through one of the larger tubes,

which is partially obstructed by a collection of mucus; sibilus is the more high-pitched whistling sound due to air passing through the smaller tubes, which are partially obstructed locally by the swelling of the mucous membrane, or by spasmodic contraction of the bronchial muscles. Hence a rhonchus may disappear on the patient coughing, and is, therefore, by no means constant in any one part, but sibilus is obviously of much graver import, and may remain in the same region for a considerable time.

Râles are produced in the bronchi, bronchioles and alveoli by (a) the bursting of air-bubbles as the air is drawn into or expelled from the lungs, and (b) by the separation of the bronchial walls from their contents during inspiration; they denote the presence of air and fluid or of viscid secretion at these points. They may be large, medium, or small, according to the size of the bronchial tube in which they are produced. Large râles are of less grave import than small, in that they imply that the larger tubes are mainly affected.

Bronchial breathing is only heard when pneumonia or collapse of lung is present, and is often associated with dulness to percussion and increased vocal resonance.

The tongue is almost invariably coated, and there are symptoms of gastro-intestinal catarrh, especially in infants and young children.

In uncomplicated cases of bronchitis of the larger and middle-sized tubes the attack usually subsides in from a week to ten days, the expectoration becoming decreased in amount and less opaque and purulent; the physical signs disappear, and convalescence is usually established within a fortnight. But in the very old and in very young children there is a greater tendency for the disease to spread to the smaller ramifications of the bronchial tubes, with liability to extensive collapse and broncho-pneumonia; this is particularly noticeable in old age, the specific fevers, measles, and whooping cough. In those sudden severe cases with marked dyspnœa and cyanosis the prognosis is always grave, and should there be any co-existent affection of the lungs, heart, kidneys, or liver the danger is greatly increased; but we have seen even these cases recover even after they appeared hopeless, and this is especially so in the case of young children.

Bronchitis may be pronounced in the early stages of typhoid fever, measles, whooping cough, which last may be indistinguishable until the development of the characteristic whoop.

(To be concluded.)

PREVENTIVE TREATMENT OF RUPTURED PERINEUM.

G. ERNEST HERMAN, M.B., F.R.C.P.

It has been pointed out that in the production of rupture of the perineum there are three factors. (1) The readiness of the tissues to tear, and their indisposition to stretch. This we cannot alter. It is sometimes recommended to lubricate the perineum, but in what way the application of grease to the epithelium can alter the structure of the muscles and fasciæ beneath I fail to perceive. There is no evidence that lubrication does good, but only the opinion of those who use it to the effect that sometimes a perineum is saved which would have been torn had it not been anointed. As it is not possible to foretell the occurrence or the extent of a perineal rupture, this opinion is not based on any substantial ground. But it is perfectly harmless to grease the perineum, so you need not be afraid of doing it. (2) The size of the child. This, if the child is to be delivered alive, you cannot alter. (3) The speed with which the child comes through the passage. This is the one factor over which you have control. The only way of preventing rupture of the perineum is by regulating this, by taking care that the perineum is not too suddenly stretched, but is given time to dilate. If you are extracting with forceps, or helping the birth of the shoulders by pulling on the head, take care to extract slowly. If the head is being driven through by powerful uterine action aided by the mother's downbearing efforts, tell the mother to leave off straining, while you press on the head so as to retard its progress. The severity of the pain which distension of the vulva causes is a safeguard against injury, for it leads the patient to cry out, and in doing so to desist from expulsive effort. Various plans have been devised to lessen the tension of the perineum. One is to press the sides of the perineum towards the centre. Try to do this when the perineum is tense; you will find your fingers simply slip over the skin without altering its position in the least. The same remark applies to the advice to press the hinder part of the perineum forwards. Putting the finger in the rectum to press the anus forwards is injurious (for you may injure the rectum) and does no good, for the perineum must, whatever its

position, dilate enough to let the greatest circumference of the head pass. Pressure directly on the perineum was the plan recommended by the older accoucheurs; and in so far as this pressure retarded the too rapid advance of the head, it may have done good. But I can imagine no other beneficial effect from this practice. Suppose a trouser so tight that the knee could not be bent without tearing it, would pressure on the knee prevent the tearing?

The above is taken from the new edition of 'Difficult Labour,' by G. Ernest Herman, M.B., F.R.C.P.; there is supplied to the medical public in this work a guide for students and practitioners to the management of difficult labour. The author in his preface points out that his threefold experience as a practitioner, as a teacher of midwifery, and as an examiner in that subject, has led him to think that a book was wanted which should give more definite guidance than is to be derived from some of the text-books of the present day. The learner finds in those volumes full details of many different things that he may do, but he seeks in vain for a clear indication of which is the best course to pursue. Dr. Herman has certainly succeeded in the fullest degree possible in telling his readers the best methods of dealing with each complication of labour, and the reasons for the directions he gives. In the present revised edition it will be found that the most important alterations are those rendered necessary by the recent advance and improvement in the technique of Cæsarean section and symphysiotomy.

This new and revised edition of Dr. Herman's book is published by Cassell and Co., Ltd. It is well printed and abundantly illustrated with 165 diagrams and photographs. A point of considerable importance is that though the volume runs into nearly five hundred pages its size is not in any way inconvenient for comfort in reading.

WE have received from Burroughs Wellcome and Co. a specimen of "Tabloid" Ophthalmic (T) Alum, gr. $\frac{1}{10}$. The process of manufacture ensures rapid and complete solubility in the lachrymal secretion. One placed in the conjunctival sac of the lower eyelid is almost instantaneously dissolved, and the solution is quickly diffused over the surface of the eye. If preferred, the "tabloid" may be dissolved in a definite quantity of sterile water.

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ABDOMINAL TUBERCULOSIS IN CHILDREN.

A Lecture delivered at the Hospital for Sick Children,
Great Ormond Street,

By GEORGE F. STILL, M.A., M.D., F.R.C.P.,

Assistant Physician to the Hospital; Assistant
Physician for Diseases of Children, King's College Hospital.

GENTLEMEN,—I propose this afternoon to say something about abdominal tuberculosis in children, and, looking at the subject from a clinical point of view, I shall consider it under two heads, namely, *tabes mesenterica* and *tuberculous peritonitis*.

Looked at purely from the clinician's standpoint neither of these is a very common condition, but if you ask the pathologist whether abdominal tuberculosis is common, he will tell you that it is one of the commonest of all tuberculous lesions.

My own statistics show that about 90 per cent., or, to be more accurate, 88·3 per cent., of tuberculous children have tuberculous lesions in the abdomen. Abdominal tuberculosis therefore is exceedingly common in the post-mortem room, but not so common in the ward; in other words, tubercle is often present in the abdomen without producing any characteristic symptoms during life. You are all familiar with this sort of abdominal tuberculosis. In examining the body of a child who has died, we will say, with symptoms pointing only to pulmonary tuberculosis, you find on opening the abdomen numerous tubercles in the spleen, a few in the liver, and probably one or two in the kidney; the mesenteric glands probably show caseous foci, and perhaps there may be one or two tuberculous ulcers in the intestine. In spite, however, of all these lesions in the abdomen there has been nothing during life to suggest abdominal tuberculosis.

Now, such a condition is of interest, it is true chiefly to the pathologist, but it seems to me that

it has also a clinical interest, for it emphasises a very important point in the tuberculosis of childhood, namely, the tendency to rapid generalisation. Whatever may have been the clinical aspect of tuberculosis in a child, whether death has been due to tuberculous meningitis or to pulmonary tuberculosis; or to any other form of tubercle, you will almost certainly find tubercle scattered more or less widely all over the body, and particularly you will find in the majority of cases some tuberculous lesions in the abdomen. Realising this tendency to rapid generalisation, we shall realise also the vital importance of the early detection of tubercle in children, for if we overlook it in the early stage we may lose our only chance of saving the child, and it is certain, both from clinical experience and post-mortem observation, that if only treatment is begun whilst the disease is still in its early stage, tuberculosis in many of its forms may be arrested, even in very young children.

The frequency of abdominal tuberculosis therefore, although it is a matter chiefly of pathological observation, has a practical significance as a manifestation of the tendency to rapid and wide dissemination of tubercle in a child.

Tabes Mesenterica.—Turning now to *tabes mesenterica*, I confess I do not much like the term; indeed, I think it would be a good thing if it dropped out of medical terminology altogether, as it has already fallen into disuse in some of the text-books. It has come to be used in a slovenly way; sometimes to indicate almost any form of abdominal tuberculosis, and sometimes—worse still—to indicate no tuberculosis at all, but simply the wasting of an ill-fed infant.

Again, it is too comprehensive a term in one sense, and too narrow in another. If by *tabes mesenterica* we mean any case in which there is tuberculosis of the mesenteric glands, we should have to include under this head nearly 60 per cent. of the cases of tuberculosis of any sort or kind, although in many of them the lesion of the mesenteric glands is of merely secondary importance, and the clinical aspect may have been that of pulmonary tuberculosis or tuberculous meningitis. On the other hand, the term is too narrow if we restrict it to the cases in which there is great enlargement of the mesenteric glands, for in this way we create an entirely artificial distinction between the case in which the glands happen to be the size

of a walnut, and the case in which, although extensively caseous, they are only slightly enlarged; and the enlargement, be it observed, is not more likely to be “primary” in the one case than in the other, so that we cannot base any distinction of “primary” and “secondary” affection of the mesenteric glands on their degree of enlargement.

It is, perhaps, most satisfactory to speak only of “tuberculosis of the mesenteric glands”; but it is convenient to use the clinical term “*tabes mesenterica*” to indicate those cases in which the tuberculous enlargement of the mesenteric glands can be detected clinically, and is associated with wasting, without evidence of tuberculous lesions elsewhere in the body. In this sense I shall use the term *tabes mesenterica*; and I may point out at once that this condition is by no means common. Tuberculosis of the mesenteric glands is common enough—59 per cent. of tuberculous children show it—but cases in which this is a prominent feature clinically are quite uncommon.

Now what is the history of such a case? The child is brought to the medical man because it has been wasting for some weeks or months, and has had colicky pains in the abdomen. The pain is generally not very severe, and, I think, is seldom sufficient to make the child cry. It is colicky in character, and recurs in an erratic way from time to time. The wasting is sometimes considerable, and in many cases the bowels have been irregular: diarrhoea has been followed by constipation, and this in turn by diarrhoea again, and so on. The appetite also varies from day to day, but is more often poor than excessive. If the temperature has been taken frequently, you will find it irregular; but, as a rule, in the early stages the temperature has not been taken, for the onset is very insidious, and the child hardly looks ill enough to be suffering from anything very serious.

The occurrence in a child of insidious wasting with colicky pain in the abdomen, especially if the symptoms have lasted several weeks, should always arouse a suspicion of *tabes mesenterica*. But this is not sufficient to establish a diagnosis; the only reliable guide in the diagnosis of *tabes mesenterica* is to actually feel the enlarged mesenteric glands; and this is by no means easy. It is comparatively seldom that one can determine clinically that the mesenteric glands are enlarged, even by the most careful palpation.

The place to feel for them is just to one side of the vertebral column; perhaps they are most easily felt a little to the left of the mid-line at or just below the level of the umbilicus. The best way is to put the child flat on its back, and pressing your two hands back on to the vertebral column, try to make the palmar surfaces of your fingers touch the bodies of the vertebræ, at the same time moving your hands as if kneading the abdomen from side to side; you may then feel the glands slip away from under your hand. Many children, however, will begin to cry at once if made to lie down on a couch, or even on their mother's lap, and for this reason I often find it best to palpate the abdomen with one hand as the child sits on my knee with the trunk bent slightly forwards.

One would think that when the glands are enlarged to the size of a walnut, or even larger, they would be easily felt, but they are not; partly, perhaps, because the child often throws its abdominal muscles into a state of rigidity directly you try to palpate, and partly because the glands are movable to some extent, and slip away to one side of the vertebral column and rest on the soft tissues of the lumbar region, where they are not easy to define. Be this as it may, in many cases where post-mortem examination shows them to be considerably enlarged, careful palpation has failed to detect them during life.

As we all know, it is not always easy to distinguish between enlarged glands and solid fæces during life, but in some of these cases of tabes mesenterica there is definite tenderness over the enlarged glands, which may help in the diagnosis; the presence of tenderness on pressure over the lump in the abdomen is strongly in favour of the mass being glands, and not fæces.

There is another method of examination which has been spoken of very highly by some observers for the detection of mesenteric glands, namely, rectal examination. In certain cases it is possible to reach glands in this way, but I can only say that so far as my own experience goes, it has not seemed to me a very useful addition to our means of diagnosis in this particular disease.

To sum up then, the symptoms of tabes mesenterica are wasting with colicky pain, irregularity of the bowels and appetite, and irregular temperature, whilst on palpation of the abdomen

enlarged glands can be felt, sometimes forming a nodular mass, and often slightly tender on pressure.

There is, however, a further important feature, which is not apparent clinically, but which, nevertheless, has a bearing upon treatment and prognosis of this condition, and may therefore be mentioned here, namely, ulceration of the intestine.

Probably even in the mildest cases and in the earliest stage of tabes mesenterica, there is some tuberculous ulceration of the bowel in nearly all the cases. My own figures, taken from examinations made whilst I was pathologist at this hospital, showed that out of 132 cases with tuberculous enlargement of the mesenteric glands, 107 cases, that is, over 80 per cent., showed ulceration of the bowel.

Ulceration of the intestine is not, of course, essential to the production of enlarged caseous glands in the mesentery. It has been shown experimentally that when small doses of tuberculous material are given to animals in their food, the mesenteric glands may become caseous without ulceration of the bowel; but judging from post-mortem evidence it seems probable that the majority of children who come under observation with tabes mesenterica have some ulceration in the intestine, and this makes the condition graver than it would be if we had only to deal with caseation of the mesenteric glands. I have seen cases, however, in which there was complete scarring of the intestinal ulcers which had been associated with caseous mesenteric glands, and it is not very rare to find calcified mesenteric glands—evidence of arrested caseation—in children.

The prognosis, therefore, of tabes mesenterica, is by no means hopeless. I have seen several of these cases improve so much that it seemed probable the disease was arrested, and if treatment is begun early, before there are signs of tubercle in the lung, and before there is any extreme wasting, I think you will find that a certain number of them will get perfectly well. It is to be remembered, however, that even if tabes mesenterica seems to have recovered completely, there are still possibilities of trouble at a later period. A mass of caseating glands may easily be the starting point of a localised inflammation of the peritoneum, and, without any more general inflammation occurring, adhesions may form

between the glands and the neighbouring structures. These local adhesions are apt to give rise to trouble long after the *tabes mesenterica* seems to have quite recovered. Not very long ago a child, aged five years, was brought to me at King's College Hospital for vomiting. He was fairly well nourished and walked up to the hospital with his mother. He had complained of some pain in the abdomen, and had been sick two or three times in the last forty-eight hours. I examined the abdomen carefully, but could feel nothing abnormal. There was, however, a look of illness about the boy's face for which I was unable to account, so I sent the boy into the ward for observation. He con-

it than by any amount of drugging; and I fancy these patients do better at the seaside than in the country inland.

Then comes the question of feeding. What one has to aim at in this respect is to give plenty of nourishing food, and at the same time to give food which shall leave as little residue as possible, which might irritate the intestine; for, as I have already mentioned, the intestine is ulcerated in most of these cases. The child should be fed as far as possible with eggs, custard, well-cooked milk-puddings, and gravy. The eggs are particularly valuable, for they contain, as you know, a large percentage of fat in the yolk. Milk and eggs are to be considered chief items in the diet,



FIG. 1.—Tubercular lymphatics passing from ulcer of intestine up to caseous gland.

tinued vomiting, and the abdomen was opened by my colleague, Mr. Burghard, who found a narrow fibrous adhesion passing from a dry caseous mesenteric gland to the ascending colon; under this a loop of ileum had slipped and become strangulated. We had a similar case in here a few months ago, the band in this case passing from the gland to the wall of the abdomen. In our post-mortem records there are other cases in which this accident occurred after *tabes mesenterica*.

As to the treatment of *tabes mesenterica*, by far the most important part is the hygienic treatment, particularly by climate. If you can send the child away to the seaside you will do far more for

and in choosing other foods, those should be selected which will leave little residue, and are not likely to give rise to fermentation in the intestine. These children are not to be starved, they require, rather, "good feeding, and plenty of it."

There is one objection which might be raised both to milk and eggs, an objection which has also been raised to the use of cod-liver oil in this condition, and that is, the blocking of the lacteals in some of the cases by caseous material. Fat, as you know, passes *viâ* the lacteals from the intestine into the lymph stream, and if this passage is blocked it may be useless to give such food.

I show you here a very beautiful specimen illustrating this point; it is a short piece of small

intestine attached to its mesentery, in which is a caseous tuberculous gland; tuberculous ulceration of the bowel has occurred, and passing up from the outer surface of the bowel over the ulcer to the mesenteric gland are seen dilated lacteals filled with whitish caseous material, perhaps, also, with some fat.

But even in such a case probably the majority of the lacteals are free from obstruction, so that the objection to fats is perhaps more theoretical than practical; at any rate I think you will find that such a diet as I have mentioned will suit these children.

As to drugs my own belief is that iodide of iron is particularly valuable in these cases. I should be sorry to have to explain its good effect, but certainly in some cases after its use the colicky pain ceases, and there seems to be general improvement. Another drug which may be useful is creosote, which is perhaps specially indicated in such a condition as this, inasmuch as not only is it an intestinal antiseptic, but it seems to have some specially beneficial influence on tuberculous lesions.

For the diarrhoea, if it occurs, I should use castor oil in small doses (mv) three times a day; it seems to have a particularly soothing effect on the mucous membrane of the lower part of the intestine, and creosote ($\text{m}\frac{1}{4}$ to mi) may usefully be combined with it.

Tuberculous Peritonitis.—As I have already said, a localised peritonitis sometimes starts from caseous mesenteric glands, and in some cases it seems likely that a generalised tuberculous peritonitis may have a similar origin. In the majority of cases, however, tuberculous peritonitis probably begins independently of any *tabes mesenterica*; indeed the mesenteric glands are found to be very little affected in some of the cases of tuberculous peritonitis which come to the post-mortem room.

Tuberculous peritonitis may be divided for practical purposes into two varieties, the plastic and the ascitic; and this division is of considerable practical importance, as we shall see when we come to consider treatment.

The plastic variety is by far the commoner; in fact, speaking roughly, I should say the proportion of plastic to ascitic is probably about ten to one.

As to the frequency of this particular form of

tuberculosis the following figures will give you some idea:—Out of 266 children who died with tuberculosis under twelve years of age, forty-five died with tuberculous peritonitis; so that 16.8 per cent. of the fatal cases of tuberculosis were cases of tuberculous peritonitis. Limiting one's observations to children under two years of age, that is, to infants, I found twelve cases of tuberculous peritonitis in 100 tuberculous infants.

This is an interesting point in view of the extraordinary statement in some of the books that tuberculous peritonitis is practically unknown in infancy. All the cases which I have included here were such as are ordinarily recognised as general tuberculous peritonitis, and most of them, if not all, had been recognised as such during life. This occurrence of tuberculous peritonitis in infancy is even more strikingly shown by the accompanying chart which shows the age incidence in each year of childhood.

The age incidence of tuberculous peritonitis as shown by this chart—which, it must be remembered, includes only cases which proved fatal—corresponds closely with that of tuberculosis in general; indeed, the chart might serve almost equally well to show the age incidence of tuberculosis in childhood. It will be seen that tuberculous peritonitis with a fatal result is most frequent in the second year of life (that is, within the period of infancy, which reaches to the end of the second year), so that so far from tuberculous peritonitis being almost unknown in infancy it is actually commoner then—at any rate as a fatal disease—than at any other period of childhood.

Trusting to one's impressions, I should have said that tuberculous peritonitis was more frequent in boys than in girls, but my own statistics show that this is not so; in 100 cases there were 48 boys and 52 girls, and in further cases the proportion came out very similar. Rilliet and Barthez, however, met with this disease in 53 boys and 33 girls. Perhaps one would be near the mark in saying that the incidence is about equal on the two sexes.

The symptoms of tuberculous peritonitis in its earliest stage are very much the same as those of *tabes mesenterica*; but later on characteristic signs appear which can hardly be mistaken.

The abdomen has a tumid appearance, with a "podgy," doughy feeling on palpation, as if it were packed full of some softish material. In

many cases there is also a transverse band-like tumour lying across the epigastrium; this I have several times seen mistaken for the lower edge of the liver, but with the exercise of a little care this mistake can be avoided, for the tumour has not only a lower edge, but also a definite upper margin—the upper edge of the thickened and caseous omentum which forms the tumour. This band-like and often slightly nodular mass lies across the epigastrium, generally just above the umbilicus, and slopes upwards towards the spleen. In some cases the edge of the liver can be defined, and is then

is often induration and redness of the skin extending outwards about an inch all round the umbilicus. This reddening and induration about the umbilicus is sometimes of value in diagnosis where the general doughy resistance is not very marked. I had a case brought to me some time ago which had been treated for rickets—the enlargement of the abdomen had simulated a rickety enlargement; but after the child had been under treatment several weeks there was noticed to be redness and induration about the umbilicus. At that stage I saw it, and it was evident by that time that we

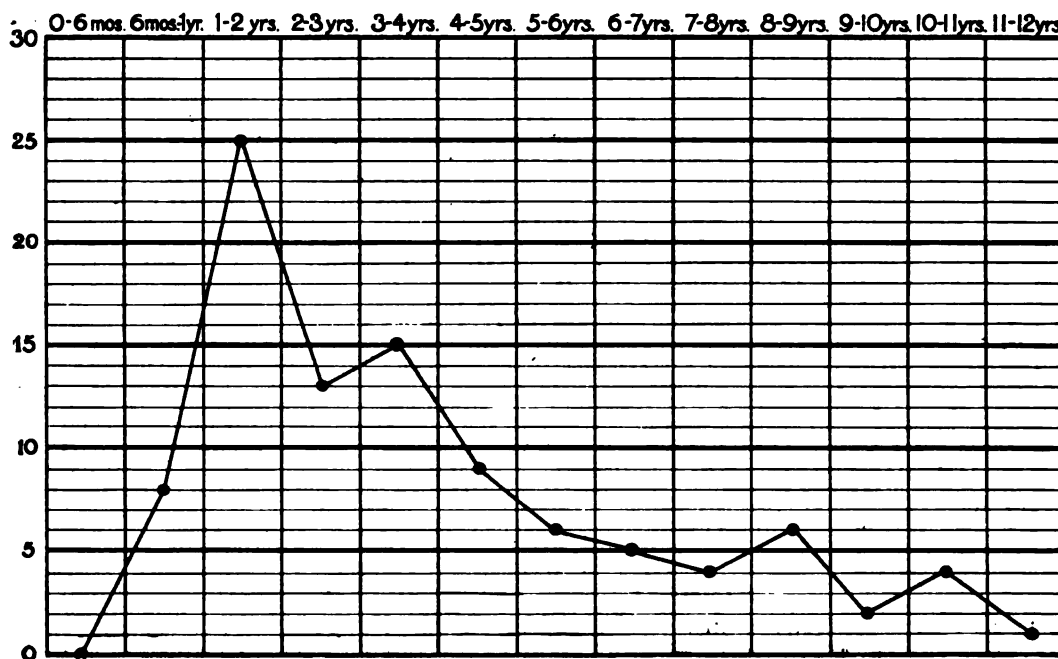


FIG. 2.—Chart showing age incidence in one hundred consecutive cases of fatal tuberculous peritonitis.

found to be quite separate from the tumour. When the abdomen is opened in these cases the caseous omentum looks very like a large pancreas lying superficially just below the edge of the liver.

The appearance of the child when the disease is advanced is very characteristic, the emaciated limbs and the shrunken covering of the chest-wall, through which the ribs stand out distinct, contrast strangely with the fulness and tumidity of the abdomen.

At this stage there is another sign which is very characteristic, namely, an unfolding of the umbilicus, which looks as if it were pushed outwards from within; and in addition to the unfolding there

had to do with a tuberculous peritonitis, which was tending to "point" at the umbilicus.

This "pointing" at the umbilicus is not very rare, and is one of the complications which must always be watched for in the course of tuberculous peritonitis. An actual discharge of broken-down caseous material may occur, and is not necessarily a matter for alarm, indeed, I have more than once known cases to do much better after this event than before it. A case which impressed this point upon me was the following:—We had a child in this hospital a few years ago extremely ill with advanced tuberculous peritonitis; the umbilicus was reddened and shiny, with induration all round

it, and it was obvious that the skin would give way very soon. I was inclined at that time to regard the prognosis as extremely bad, and when the parents insisted on taking the child away from the hospital they were strongly dissuaded from doing so on the ground that it might die on the way home. What actually happened was that they took the child out against our advice; a day or two later the skin over the umbilicus gave way, and there was a free discharge of caseous debris; and when we subsequently heard of the child, about six weeks later, it was running about at home, apparently making good progress towards recovery.

A much more serious accident, and one which is usually speedily fatal, is perforation of the bowel before there is much adhesion. In such a case the symptoms may suddenly change from those of tuberculous peritonitis to those of acute septic peritonitis.

When there is already much adhesion, perforation is not so serious an occurrence; for then a localised abscess forms, which may track amongst the coils and eventually point at the umbilicus or elsewhere, and in spite of a fæcal discharge from the abscess the child may seem little the worse. Perforation does not always take place from the inside of the bowel by rupture of the peritoneum over tuberculous ulcer. In many cases a deposit of caseous material between two coils breaks down and discharges into the neighbouring bowel, so that the perforation occurs from without.

Ulceration of the bowel is present in many—about 70 per cent.—of the cases. I found it in fifty-four out of seventy-seven cases where it was possible to examine the bowel sufficiently thoroughly.

An unusual complication which I have seen in the early stage of tuberculous peritonitis is acute distension of the abdomen, a misleading symptom, as it suggests rather some acute septic peritonitis than a tuberculous condition, indeed the diagnosis in such a case is hardly possible until the gradual subsidence of the distension, and the increasing doughy resistance of the abdomen makes the tuberculous nature of the peritonitis clear.

In the ascitic variety the symptoms are very different; here one finds the abdomen distended with fluid as in other forms of ascites, and it is often quite impossible at first to make a diagnosis. If one can have the abdomen tapped, and a

bacteriological investigation made, it may be possible to determine the variety of ascites, but in most cases it is necessary to wait some time, perhaps several weeks, before a positive diagnosis is possible.

In such a case, always examine the child thoroughly all over, and see if you can find any evidence of tubercle elsewhere. I saw a case not long ago in which the abdomen was distended with ascites of doubtful nature. On examining the child—a boy about five years old—I had almost come to the conclusion that there was nothing to throw any light on the ascites, when it occurred to me as a matter of routine to examine the testicles; I found the epididymis on one side considerably enlarged and hard, making it practically certain that the ascites was tuberculous.

These children rarely die with ascites. As a rule the fluid gradually disappears, and as adhesions form, the condition becomes that of the ordinary plastic form. This is a point worth remembering, the mere disappearance of fluid, whether by incision or by tapping or by less active treatment, does not show that the peritonitis is cured, it may still run the course of a plastic tuberculous peritonitis, and it would be premature to boast of a cure until several months have passed, and until you can be quite sure that you have not simply converted a case of ascitic tuberculous peritonitis into one of the plastic variety.

The prognosis of tuberculous ascites is good so far as the immediate present is concerned; but if the ascites is gradually replaced by general matting and adhesions, as it is in many cases, the prognosis does not differ from that of cases in which the peritonitis is plastic or adhesive from the beginning.

The prognosis of tuberculous peritonitis in children is by no means always unfavourable. It is difficult to determine the proportion of recoveries amongst hospital patients, for the reason that so many pass out of observation after a few weeks or months, and their subsequent course is unknown; but certainly many cases leave the hospital greatly improved, and some apparently cured.

I say "apparently cured," for I would insist very strongly on the need for caution in talking about "cure" or "recovery" in such cases; the case which I now show you may illustrate this point.

This little boy, aged about six years, was brought

to me at King's College Hospital nearly two years ago for wasting and pain in the abdomen. There was some resistance, and impairment of note on percussion over the lower part of the abdomen, and it seemed quite certain that he had tuberculous peritonitis; the diagnosis was confirmed by the scar of an old tuberculous dactylitis on the right hand.

Under medical treatment the resistance gradually disappeared, and the abdomen became as supple as a normal abdomen.

But to call such a case cured would, I think, be quite unfair. It is true that about eighteen months have elapsed since the abdominal symptoms and signs disappeared, and perhaps the tuberculous peritonitis is cured, but his tuberculosis is not cured. During the last few weeks the supracondylar gland in the right arm has caseated and broken down, and now he has occasional colicky pains in the abdomen, which suggest fresh mischief, or, possibly, old adhesions of the peritoneum.

Unless a case has been kept under observation for at least a year or eighteen months after the supposed recovery, it is worthless as evidence of a cure, for some at least of the so-called "recoveries," both after surgical and after medical treatment, end fatally within two or three years with other manifestations of tuberculosis or a recrudescence of the abdominal disease.

Unfortunately, in many cases there is not even a temporary improvement, and in a series of fatal cases in this hospital I found that the average duration was about five months; the shortest was four weeks.

The question of the frequency of recovery with various methods of treatment is of great practical importance, for of recent years we have heard much of the surgical treatment of tuberculous peritonitis. I do not propose to discuss this question here, but I may perhaps be allowed to say that in my opinion any surgical interference with tuberculous peritonitis is to be adopted only with the greatest caution; and that whilst I believe there are cases, particularly of the ascitic form, in which operative treatment may be valuable, I consider the use of laparotomy in the plastic form of tuberculous peritonitis as rarely if ever advisable.

In the medical treatment of this condition, climate stands first and foremost; get the child away to the seaside or the country as quickly as

can be managed, see that it is taken out in some sort of carriage in the open air daily, and almost all day if possible. I fancy that a bracing atmosphere suits these children best, at any rate in the summer, but cold is to be avoided; and a dry gravel soil is to be preferred to sand or clay. Good feeding is essential, and must be upon the same lines as in *tabes mesenterica*; in both conditions the ulceration of the bowel is to be remembered.

Rest in bed combined with good feeding is probably responsible for the improvement in many cases admitted to hospital, and in the case of the poor, the difficulty of getting proper food often makes it advisable to admit the child to hospital; but apart from this, I believe that for most cases, except in the advanced stage of the disease, a stay in the country is of more value than a stay in hospital.

As to drugs, the time-honoured application of unguentum hydrargyri may do some good, but I confess I am very doubtful of its efficacy. Dr. Burney Yeo has spoken highly of the application of iodoform, for example, in the unguentum iodoformi, and from some experiments which I made with this preparation I satisfied myself that its application to the abdomen was followed by the very rapid appearance of iodine or iodide in the urine, and one can well imagine that carried thus in the blood, the drug may have some influence on the tuberculous process in the peritoneum. Internally I am inclined to give extract of malt, or syrupus ferri iodidi, and sometimes creosote, especially if the stools are particularly offensive; but whatever improvement occurs—and I have again and again seen improvement occur after the use of some of these drugs—it is difficult to know how much to attribute to the drugs, and how much to the improvement of hygiene in the matter of fresh air and food.

BAMBERGER describes the case of a man who apparently had never wept from the left eye. A small opening was observed lying 3 cm. to the outer side of the eye, into which the sound could be passed for a distance of half a cm. About every ten minutes a tear-drop appeared at this opening and rolled down the cheek. It appears to be an abnormal opening connecting with the left lacrimal gland.—*Philadelphia Med. Journ.*, Nov. 16th, 1901.

A CLINICAL LECTURE ON CASES OF SKIN DISEASE.

Delivered at the West London Hospital,
February 11th, 1901.

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GENTLEMEN,—The first case I want to show you is an interesting one of morphœa, a somewhat rare complaint. It commenced in this woman about a year or fourteen months ago, and she first came under my notice in the Blackfriars Hospital about six months ago. She then exhibited a white sclerosed patch on the cheek on the left side; the skin could not be pinched up over it, being hard, like a piece of parchment or white leather; there was a history of her having received a blow on the cheek some six months or so before coming to the hospital. This patch had spread and extended to the eye, and had a white parchment look with a rosy-ringed edge; since that time the condition has improved very much, and the patch is now disappearing, indeed, at the present time, its centre shows reddish islands. I gave her thyroid gland in five-grain tabloids, one to be taken every night, and I gave her some simple ointment to be rubbed into the spot, recommending slight massage for the affected part. I cannot say that the results of treatment in morphœa are usually satisfactory, but the records show that some cases have done well under treatment, and that a good many have spontaneously recovered in the course of time. This case was particularly interesting to me because it followed two or three others in which there was also a history of some antecedent nervous disturbance. One of these, a girl, had a large patch over the shoulder which was about ten inches in diameter; she gave a history of having fallen down some steps. In that case, too, recovery took place under treatment. The pathology of these cases of morphœa—as we call the affection when localised and comparatively slight—is just the same as that of the more extensive cases of true sclerodermia, as it is called where the patches are thick and extensive. About a year ago, I showed a man here who had generalised sclerodermia—large, thickened, hide-bound patches over

the body and limbs—he is now practically well. Under the microscope the connective-tissue elements of the skin are seen to be greatly hypertrophied. As a result of this increase, the other organs of the skin, the follicles, the blood-vessels, etc., become atrophied. At first there seems to be some slight inflammation; leucocytes appear in abundance around the vessels, and there is a general œdematous and swollen appearance; after a time a shrinking of the new connective tissue takes place, to which the parchment-like appearance is partly due; the level of the sclerosed area is then somewhat below that of the skin around. Another point of interest in this case lies in the fact that the patient is very neurotic, and it is quite possible that her nervous temperament may have some effect as a predisposing cause of the complaint. She had an abdominal operation performed on her some time ago, and ever since then she has been somewhat out of sorts, and she attributes all her troubles to this operation. It is also noteworthy that recently she has been troubled with deafness and a discharge from the left ear, *i.e.*, on the same side as the morphœa patch; I sent her to Dr. Ball's department, but I have not yet heard the result of his examination.

2. The next case is one of ordinary impetigo, a very common complaint in this part of London. The patient, a little girl, came to the hospital two days ago with her face covered with crusts. Her mother says she is already very much better, and in a few days the crusts will entirely disappear and she will be all right. I ordered her ammoniated mercury ointment to be applied after bathing with some antiseptic wash to soften the crusts. I prefer this treatment to that of poulticing, because the latter may aid in the cultivation of the staphylococcus, which is the cause of impetigo. Such cases as this, with the face masked or covered with crusts, used to be called "porrigo larvalis." The present appearance of this case, after only two or three days' treatment, shows how easily the complaint can be dealt with.

3. This woman came here with a puckered sore on the side of the nostril, which she has had since last November. Its appearance points at first sight to rodent ulcer, but on carefully feeling it you will find that it is not so hard and gristly as it ought to be if it was a case of rodent ulcer. That fact made me ask her if there was anything else the

matter, and she told me there was "something" inside the nose with a discharge, and then I found internal ulcerations as well. The appearance of the tongue in this case was also very specific, with scarring and superficial ulceration both on the sides and dorsum. She has had one miscarriage, but there is no other specific history to be obtained. I put her upon iodide of potassium, five grains three times a day, and applied externally to the sore some ammoniated mercury ointment. If the condition of the nose be, as I believe, syphilitic, rodent ulcer would even then be not precluded, for it occasionally develops upon a syphilitic base; but we shall soon see, in the course of the next few weeks, whether she improves on iodide, and if I then come to the conclusion that we have to do with rodent ulcer, I shall either treat her by means of the Roentgen rays or else excise freely. At the present time these two procedures are, I think, the best for the treatment of rodent ulcer. I have recently seen one or two extremely good results following the Roentgen-ray treatment, the rodent ulcer softening down and finally disappearing, leaving only a healthy piece of scar tissue. Dr. Sequeira has treated one or two cases with great success at the London Hospital, and other cases have been reported with satisfactory results. In connection with cases of rodent ulcer, I would add that anyone who has had much experience of the disease ought not to have the least doubt about the diagnosis as a general rule, because the hardness of the edge of the growth is such an unmistakeable and typical character. Every now and then, however, one does come across cases which are doubtful. Quite lately I have seen three cases in which I could not at first say definitely whether they were rodent ulcer or not. Lupus and nodular ulcerating syphilides are most likely confused with it; the new growth of the former is always much softer than that of rodent ulcer, and the raised edge of the syphilide is intermediate in hardness between the two.

4. This little boy has a condition approaching ichthyosis, with a very rough and dry skin, especially on the exterior surfaces. In this country we call such cases "xerodermia," but on the continent they make no distinction between "xerodermia" and "ichthyosis," the former being considered as merely mild forms of "ichthyosis." It is only fair to say that there are cases of eczema which simulate in some degree xerodermia, and the

diagnosis at first is not always easy. As a rule the dryness of the skin indicating xerodermia and ichthyosis is practically congenital, or appearing in early life, and it is less readily relievable. This patient's skin is getting normal in parts owing to treatment; indeed, the xerodermic appearance has, in some parts, almost disappeared. The skin never itched much. Such cases are very liable to develop ordinary eczema about the flexures, eyelids, etc.

Soon after thyroid gland was first introduced in dermatological therapeutics, I came to the conclusion that it was of far more benefit in xerodermic cases than in psoriasis; and I now very often put such patients on small doses of thyroid gland. I rely, however, more on external treatment—frequent tar baths, followed by emollient inunctions. This patient is having a tar bath every day, is rubbed morning and evening with an ointment containing a little creolin, and is also taking one or two lozenges of thyroid gland per diem. In France they have had great success in cases of ichthyosis with free applications of glycerine. I generally use creolin or some sort of tar preparation with the glycerine in lotions or baths, e.g. two drachms of glycerine and half a drachm of creolin to ten ounces of water to form the lotion. For the bath I recommend a tablespoonful of glycerine and a teaspoonful of creolin to every six gallons of water. The subsequent application of some emollient ointment makes the skin, in time, still more soft and flexible and comfortable. If the case is an extreme one, the condition may be serious, inasmuch as the skin being so hard, the use of the joints is interfered with, and painful cracks and sores may be produced. The thyroid gland given internally seems to have some effect on the nutrition of the skin, and possibly on the sweat glands and proper secretions of the skin. But I must warn you only to order thyroid gland with very great care, and always to watch the case in which you have prescribed it; because you may have disagreeable symptoms of thyroidism set up, such as palpitation, neuralgic pains, etc., if the patients take too much, or if its use is continued for too long a time.

5. This girl I now show you has had falling of the hair in large patches for the last four years; the hair has come off, and then it has grown again, and then fallen off again. There is no history of

seborrhœa, but one of constant severe headaches. It was for a long time the view in this country and in Germany that alopecia areata, of which this case is an example, is caused by some neurosis, and that headaches are often the forerunners of the falling of hair. We have gone into the question carefully for some years past, and we have come to the conclusion that in the majority of the cases that is not the usual course of events. The French have always had some suspicion that there was some microbe at the bottom of these cases, and it has been now proved by Sabouraud to the full acceptance of most dermatologists that a microbe is the principal cause of alopecia areata, and that it is very similar to the micro-organisms which are connected with seborrhœic and acne lesions. We now hear of a good many cases following seborrhœa; the scalp being dry and scurfy for some time before the hair falls out, and the hair coming out suddenly in patches without any previous great change. This case is interesting in being an exception to the ordinary rule, for generally these cases do not give a history of headache. In all these cases the same treatment is efficacious, namely the simultaneous application of a germicide ointment and some stimulant. I have adopted at this hospital the same method which has been for so many years successfully employed at Blackfriars, viz. rubbing in once every ten days or fortnight a very strong cantharides solution; we use Burt's fluid there as being rather stronger and less unpleasant in smell than the liquor epispasticus of the British Pharmacopœia. In using it, I dip the brush into the liquid and take up as little as I can, and then rub very hard into the patches and all around them; the object being to try and get some of the fluid into the hair follicles, and desquamate all the epidermis from the mouth of the hair follicles. This should remove the baccilli, which are usually to be found near to the mouth of the follicle, above the orifice of the sebaceous gland. The cantharides solution, moreover, acts as a general stimulant, increasing the flow of blood, and stimulating the nutrition of the hair. The germicide ointment that I have rubbed in every night contains salicylic and carbolic acids, of each 3j to the ounce of vaseline. I do not as a rule give any medicine internally unless it is indicated for some other reason. To this patient

I am giving an alkaline bitter and aperient mixture. I have no doubt that she will get her hair back.

6. This boy is an instance of extensive ringworm with impetigo at the same time. Certain authorities look upon the coexistence of these two diseases as a very rare thing, but this is not our experience in these parts. If you look with a lens, and scrape away the crusts, you will find numerous short thick diseased hairs growing irregularly, the whole scalp being implicated. He will be at first treated with ammoniated mercury ointment, which will cure the impetigo, and before this is applied he will have the scalp bathed with a warm antiseptic solution to soften the crusts. In a few days another ointment, containing carbolic and salicylic acids, one drachm of each to the ounce, will be used for the benefit of the tinea condition.

7. Here is another case of xerodermia, and again you have the same characteristic dry skin with defective perspiration. You can feel on the front of the legs that the follicles are enlarged, reminding one of a nutmeg grater. The boy also has some in the flexures of his joints. He is using our compound creolin ointment after bathing, with a lotion containing one drachm of creolin and two of glycerine to about a pint of water. The ointment contains creolin 3ss, Hydrarg. ammon. gr. x, and vaseline one ounce.

8. This man had his condition a month before I saw him. It is interesting inasmuch as there was some uncertainty as to whether it was a case of lichen planus or papular eczema. At first sight, it seems rather curious that one could mistake the two diseases; but the copious eruption was crusted and moist in places like an eczema, and very irritable, and elsewhere the colour and the glistening of the papules indicated lichen planus. You may get lichen planus in all parts of the body, but the most common positions are on the front of the shins and forearms. It is a most irritable disease, pruritus being very marked indeed; the itching is now better. He has been freely applying a compound creolin ointment after bathing in weak creolin bath. The patient has improved very much since he has been under treatment.

9. This is a case with a history of six months' duration. You see large furuncles on the legs.

He had also great big boils in various other places. He was admitted only on February 5th, and the improvement in the short time which has elapsed is most marked. The fact is furunculi can generally be easily cured by freely bathing with some antiseptic lotion first and afterwards applying a compound sulphur ointment, such as we use at Blackfriars; it contains ten grains of ammoniated mercury, twenty-five or thirty grains of precipitated sulphur, and ten grains of sulphide of mercury to the ounce of vaseline. Before the ointment is applied the skin all round should be washed with hot water and some antiseptic lotion. I often use creolin, especially at the hospital, because it is both inexpensive and sufficiently efficacious, but boric acid, sublimate, or carbolic acid will do just as well. Sometimes a condition of furunculosis may develop which may last for a very long time; staphylococci, indeed, seem to get right into the system, and the boils may break out anywhere. The patient in this case is in good health otherwise; but often there is constipation and digestive troubles, which must be treated as indicated.

10. This is an inpatient of the hospital who was quite well up to May last when she first noticed round patches on the head covered with white scales. In July a scaly eruption appeared behind the knees, in the bends of the elbows, and in the groins, then they spread all over the back, chest, legs, and arms. She attended the Blackfriars Hospital for some time, and three weeks ago she was admitted here. She has never had anything of the kind before, nor have any members of her family had any skin disease. It is a case of severe psoriasis, which has become universal, and may now be considered a general exfoliative dermatitis. The irritation has been very great, and as we can see, the skin everywhere is much inflamed. Certain text-books lead one to suppose that you never get psoriasis except on the extensor surfaces; this, of course, is a mistake, and is shown to be so in this case. The eruption, indeed, is not infrequently situated on the abdomen and chest; and although the extensor surfaces, especially the knees and elbows, are the most favourite positions for it, I have had cases in which it has never appeared throughout its whole course anywhere except on the flexor surfaces. Occasionally, as in the present instance,

we meet with cases of psoriasis which become transformed into general exfoliative dermatitis, and these are very serious. This patient has improved very much since admission; she has a tar bath every morning, the bath containing one drachm of creolin to six gallons of water—she is kept in the bath for half an hour—and free inunction with a strong creolin ointment, containing one drachm of creolin, ten grains of ammoniated mercury, and twenty grains of salicylic acid. Recently I have put her on some salicylin. For any obstinate patches, which may remain on the legs and arms, I shall probably use a pigment containing chrysarobin, forty grains to the ounce of liquor guttaperchæ, to be painted on. It is very difficult to distinguish psoriasis, which has taken on this form from pityriasis rubra.

11. The next case is a very serious and severe one of universal dermatitis herpetiformis in a woman eighty years of age. Dermatitis herpetiformis is a very interesting disease which used to be confounded with pemphigus. It is characterised by patches of erythema coming out in a herpetiform way in groups, and upon these are developed vesicles or papules, or, more commonly in our experience in this country, blebs, some of which may be as big as a hen's egg. The fact of its occurring in these herpetiform groups, the general health being very often very much affected, and the fact that there is extreme pruritus or burning itching, together with its great obstinacy to treatment, point to its being a distinct disease from ordinary pemphigus. This patient was quite well up to the end of May last, when small red specks were noted first of all on the chest and arms. In pemphigus, the general thing is the immediate appearance of blebs without inflammation, whereas in dermatitis herpetiformis you nearly always, at some time or another, have patches of erythema. Three months later the eruption had spread to the buttocks and legs. She says that most of the spots after a day come up into blebs and break, discharging a white substance and leaving a sore place. The portion of the body first affected is to some extent healed, and the irritation is not nearly so severe as in those parts of the body where the eruption is more recent. My house physician, Mr. Last, has furnished the following very excellent note of the case:—"The patient has never had anything of the kind previous to last

May. She was born in the country, and lived there for the first twenty years of her life; since that time she has lived in London. She was married at thirty-eight, and has had neither children nor miscarriages. On admission, she was pale, anæmic, poorly nourished, and feeble, she complained chiefly of the extreme itching. With regard to the cutaneous condition, the following different stages may be noticed: (1) patches of hyperæmia of a dusky red colour, gradually fading to the colour of the normal skin; (2) blood crusts are seen in a few places, surrounded by areas of hyperæmia; (3) a few hyperæmic patches appear to be raised above the general surface, and show through the transparent cutis; (4) vesicles of different shapes and sizes, varying from that of a pin's head to a small acorn; some of the vesicles are surrounded by an area of hyperæmia, others are not; the vesicles are unilocular and contain clear serum; (5) shallow ulcers; these are of different sizes and shapes, varying from the size of a threepenny bit to the size of a half-crown or even larger. The margin of the ulcers are sinuous and irregular, but sharply marked off from the area of hyperæmia which surrounds them, and they are not raised above the general surface of the skin, nor are the edges rolled over. The surface of the ulcer is smooth and covered with what looks like very fine granules, although some of them present a glazed appearance. The surface is only very slightly depressed, and in many instances not at all, but where it is the edges are sharply cut. The base of the ulcer is scarcely infiltrated at all. The ulcers as a whole show no tendency either to heal or to spread. One of the vesicles has recently burst, the torn dry edges are seen arranged round an area of hyperæmia. With reference to distribution, the hyperæmia is found all over the legs and arms, chest and back. The largest of the vesicles are seen on the feet and legs, the smallest on the chest. The largest ulcers are seen on the buttocks, and in the neighbourhood of any point where pressure is brought to bear. There are very few ulcers on the chest and abdomen as compared with the buttocks."

The patient was very weak after her bath this morning, and was given brandy. I should also mention that just before the affection appeared, she had a slight stroke of facial paralysis on the right side. With regard to treatment, few cases of

this disease do well with arsenic. Dühring, of Philadelphia, who first called attention to this peculiar group of bullous diseases, has been followed by most other dermatologists in accepting it as a definite type, and one point on which he laid special stress was the extreme difficulty in treating such cases. He found tar applications of use, but does not think arsenic is much good; sometimes he gives these patients iron, and salicylates have in some cases been beneficial. The skin, I should tell you, still itches very much. Another distinction between dermatitis herpetiformis and pemphigus lies in the fact that if the base of the ulcers in the former are scraped, corpuscles are found which are markedly stained by eosin; this has been noticed by other observers. These eosinophyll bodies are also found in the blood.

[Since the lecture was given the patient had another apoplectic attack, to which she succumbed.]

12. This man has several large blisters underneath the feet, as well as in other places. It is a case of ordinary pemphigus. You will notice small blisters on the right foot; they prevented the patient from walking, and so I took him in here. His general health is otherwise pretty good, although he, too, complains of itching.

13. This man was a pavior, and whilst doing some work in connection with sewers he suddenly got very ill, and came to the hospital with his feet covered with masses of ulceration and enormously crusted, considerable ulceration of the mouth, and a sort of fungoid granulation from the lips and tongue. He had also in the groins large fungating masses which looked like chondyloma; my first opinion of the case was that it was a severe case of old tertiary syphilis, but, looking more closely, I found one or two blebs which put me on the right track, viz., that it was a case of that very rare form of pemphigus—pemphigus vegetans, of which there are only a few cases yet recorded. Mr. Pernet, the pathologist at the Blackfriars Hospital, found in the blood some micro-organisms, of which he has made cultivations. There is no history of syphilis, but he states that he had a bad throat which lasted about four months, when he was working in the sewers. About nine weeks before admission he noticed one or two blisters appearing on the chest, and at the same time he complained

of severe itching and burning sensation, swelling of the head and tongue, with pain in the back of the head, and great weakness. He went to bed, being unable to continue his work. Blisters then developed in the left groin, and a week later on the head. The doctor who saw him at this period considered the case one of erysipelas. Next the right buttock and left foot were affected. Since his admission, blisters have appeared on the front and back of the left arm and on the back of the neck; also in the right groin and scrotum. The patient unfortunately declined to remain in the hospital, and I have since learnt that he died very soon afterwards.

ON THE CAUSATION, SYMPTOMS, AND TREATMENT OF ACUTE AND SUBACUTE BRONCHITIS.

By J. COURTENAY MACWATTERS,
M.R.C.S., L.R.C.P.

(Concluded from p. 111.)

Treatment.—In mild cases of "cold on the chest," little is needed beyond ordinary household remedies, as a mustard poultice or turpentine fomentations to the chest, a hot-water and mustard foot bath, and a diaphoretic draught, such as Sweet Spirits of Nitre, Liq. Ammon. Acet., or a Dover's powder at bedtime. The second of these is the most suitable for repeated administration; but the first acts more quickly, two teaspoonfuls in water, taken as soon as mixed, rarely fails to produce diaphoresis. If the patient cannot remain indoors for even a day or two it is advisable to avoid the diaphoretic, which would render him more susceptible to cold and involve the risk of converting a simple bronchial attack into a severe bronchitis of the finer tubes. With this in view I consider that a Turkish bath, so favourite a remedy with many people in the initial stages of a "cold on the chest," is attended with risk, as it can seldom be procured without the patient being subsequently exposed to cold.

We have seen in considering the course of the disease, that as soon as the mucous membrane begins to secrete, the patient feels better; the increased rate of breathing and dyspnoea disappear.

The chief indications should, therefore, be to reduce the temperature when raised, and to bring about a speedy induction of secretion from the inflamed mucous membrane.

The signification of the early dry harassing cough must be interpreted before treatment is ordered. We saw that the initial stage was due to hyperæmia, with concomitant dryness and swelling of the bronchial mucous membrane, and with this in mind the correct indication is to reduce this hyperæmia and to allay, if not entirely prevent, what is both useless, distressing, and exhausting. Such drugs as will exert a depletive action on the vascular supply to the bronchi—practically amounting to an indirect bleeding—will obviously do the former, whilst the initial cough may be combated by sedatives, as suggested later on. If one can only get the case early the nitrites or Liq. Trinitrin in $\frac{1}{4}$ -minim doses every two hours act well, and I believe cut short, or at least modify the subsequent course of the attack. Other drugs more extensively used act to some extent in the same manner; some of the almost brilliant results occasionally seen following the administration of aconite in children, I believe to be due to this general vaso-dilator action. Antimony is a most useful drug in this early stage, acting, as above mentioned, as a depletive to the engorged mucous membrane, promoting secretion and reducing fever. Large doses should be avoided, as depressing, and if there is cardiac weakness antimony had better be withheld altogether. Antimonial preparations have the advantage of causing secretion from the bronchi, but have the disadvantage of provoking cough, unless combined with some sedative as Tr. Camph. Co., or Spts. Chloroformi. The induction of secretion by the inflamed mucous membrane can most rapidly be brought about by the administration of alkalies. It is well known, both clinically and physiologically, that alkalies produce a bronchial secretion, or when already present increase the proportion of water in it, and so render it thinner and more easy of expectoration. Direct antacids are apt to irritate the stomach in large doses, and should, therefore, be avoided. We therefore have recourse to indirect antacids, the best of which are Citrate of Potash and Acetate of Soda or Potash.

Ipecacuanha is similar in its action to the alkalies, but has the disadvantage of being nauseating

in adults, even in 10-minim doses, unless the patient is cyanosed; it also provokes cough.

The combination of Spirits of Chloroform mxx to mxx , with Morphine gr. $\frac{1}{10}$, or Codeine Phosphate gr. $\frac{1}{4}$, will give great relief, both from the cough and the pain; but obviously it is not unattended with risk, and such sedatives should be employed with the utmost care, and only when the patient may be constantly observed, and of course should be at once withheld when sputa appear.

The spasmodic character of the cough in the first stage is probably due to spasm of the bronchial muscles. Here, again, I have found Liq. Trinitrine in oft-repeated doses of $\frac{1}{2}\text{m}$ act well, but it is apt to produce headache if continued. I have found great relief obtained from a 5-gr. tabloid of caffeine citrate, as advocated by Skerrit; its apparent uncertain action he thinks to be due to the fact that a certain proportion of the dyspnoea is due to a narrowing of the lumen of the tubes by swelling and adherence of thick mucus, and secondly, by spasmodic contraction of the bronchial muscles; caffeine relieves the latter, but does not affect the former, and so acts best in combination with the alkalies.

While the mucous membrane remains dry and there is no sputum, steam inhalations are useful in allaying cough and the tightness and rawness of the chest. Tr. Benzoin. Co. 3ss to 3j, or Tr. Belladonnæ, added to a pint of boiling water, yields an inhalation that some patients find most comforting, or a "steam tent" may be erected round the cot in the case of a child, and it is during this stage that a bronchitis kettle will be found beneficial. It is sufficiently obvious that when secretion is liquid and plentiful, the presence of an atmosphere laden with moisture must only aggravate the trouble.

A mustard leaf will soon relieve the tightness and rawness of chest in most cases, but in more severe ones, we must resort at the onset to large well-made poultices of linseed meal, with a tablespoonful of mustard in the first one or two applied to the front and back of the chest, the mustard being omitted as soon as thorough redness is produced.

Purgatives prove most useful, relieving the usual accompaniments of the early stages, hepatic congestion, gastric catarrh, and constipation. A smart mercurial purge, as Pil. Colocynth Hyos-

cyamus and Calomel or Hydrarg. Subchlor., gr. iii to iv, followed some six hours later by a saline draught, will generally prove effectual.

In any but very slight cases the patient must obviously be kept in bed till the temperature drops. The room should be large, and should be kept at a temperature of from 62° to 65° F. The diet should consist of warm fluids only—milk, two pints, and broth, beef tea, or Bovril, one pint, during the twenty-four hours, given in five-ounce feeds every two hours, for which the patient should be awakened if sleeping long. Cough and expectoration should be encouraged as soon as sputa appear, and in elderly subjects change of position should be frequently insisted on, to prevent the accumulation of sputum in the tubes at the bases. Very hot drinks of lemon water will allay cough and relieve thirst. A tumbler full of boiling milk, cooled down with soda water or "Apollinaris," and more especially if a pinch of bicarbonate of soda be added, proves a most soothing and comforting drink to many, assisting expectoration, and relieving the "tightness of the chest."

During the early stages brandy given in three or four teaspoonful doses in hot milk is useful, and in severe cases one to two drachms for adults, and 5 to 10 minims for children, every two hours, may be required. The value of brandy in severe cases, especially in young children, cannot be overestimated.

As the case proceeds and expectoration is established, stimulant expectorants may be combined with or replace the alkalies. Carbonate of Ammonium, gr. $\frac{1}{2}$ to 2, Compd. Tincture of Benzoin, 3ss, and Infus. of Senega, are most useful, while later Squills, or Compound Tincture of Camphor, are comforting.

If expectoration is profuse, and does not seem to diminish, the tincture of Virginian prune (3ss—3j) and Tincture of Belladonna (mv — x) will give good results, with which may be combined Syr. Picis Liq. (3ss to 3j), when the attacks show any tendency to become chronic.

During the early stages of bronchitis, the accompanying nasal catarrh at times proves most troublesome. Great relief may be obtained and sleep procured by the frequent use of a nasal spray, such as the following:—Menthol, gr. x, Cocaine Alk., gr. xx, Eucalyptol, gr. x, liquid

paraffin, ad ʒj, Misce, to be used in one of the many excellent "Atomisers" now to be obtained. The initial harassing cough may be to a great extent relieved by pastilles of menthol and coccaïne, or, if the throat be sore, the addition of rhatany or red gum to the pastille is grateful. In grave cases, where the patient's strength is failing, expectoration imperfect, and the secretions accumulating in the tubes, I have found the frequent administration of strychnine, in doses of gr. $\frac{1}{80}$ to $\frac{1}{30}$, hypodermically, certainly assist the clearing of the air-passages, and I have no doubt this may in some part be due to an action of the alkaloid on the bronchial muscles themselves.

Oxygen has frequently been used in cases of bronchitis where there is much cyanosis and a failing heart, and we have often seen relief from its use. The gas should be always given warm and moist, by causing it to bubble through hot water before being supplied to the patient.

During the second stage of bronchitis, where there is a varying amount of secretion, some care is necessary in the choosing of the most suitable drugs to act as astringents, for some act more on the water than the other constituents of the secretion, the result of which is that the sputum, though less in amount, is rendered more difficult of expulsion, and so cough is increased. Ammonium Carbonate and infusion of senega, as above mentioned, are valuable drugs in reducing the secretion, but they at times produce this undesired effect. The addition of Ammonium Chloride in 15 gr. doses every four hours will speedily render the sputum less tenacious. This salt has the disadvantage of an unpleasantly saline taste; but this may be covered, at least to some extent, by Chloroform water or Syrup. Tr. Euphorbiæ Pilulif., in doses of from 10m to 30m will ease the spasmodic nature of the cough in asthmatical cases, but is apt to produce nausea if given in too large doses, or over too long a period.

Santa Yerba, or the Sacred Herb of California, has been for centuries used by the natives of Central America as a remedy for coughs. I have found the liquid extract extremely beneficial in the later stages of bronchitis. It is said to reduce the watery and mucous constituents in equal proportions, and consequently does not render it more difficult of expectoration. It has been used to cover the bitter taste of quinine, and contains a

gum resin that is precipitated by the addition of water, making an unsightly mixture. This may, however, be avoided to some extent by the addition to the mixture of some spirits and alkalies, as Sp. Chloroformi, Sp. Ammon. Aromat. or Sodæ Bicarb.

Iodide of Potassium is more particularly indicated in gouty cases, relieving spasm as well as lowering blood-pressure. It increases the amount of water in the secretions of the bronchi. I have found this drug act like magic in those cases of asthma simulating bronchitis in children. A dose of 2 to 3 grains repeated every two hours for four doses, or till relieved, being attended with the most gratifying results in the case of a child æt. 4; the cyanosis, rapid breathing, and cough being very quickly relieved, and being quite unattended by any signs of depression.

Copaiba acts well in relieving the cough at the end of an attack of bronchitis, especially that annoying cough with little expectoration that none of the ordinary expectorants will get rid of. It forms an unsightly mixture, owing to the presence of the gum resin; but this may be got rid of by presenting it in capsules, or giving the patent mixture known as "Sanmetto" in teaspoonful doses three or four times a day.

Caffeine I only refer to again, to point out that the very troublesome cough a patient recovering from a mild attack of bronchitis will complain of on retiring to bed—to a cold room—will yield to a 5-gr. tabloid of the Citrate at bedtime. This cough being no doubt due to spasmodic contraction of the bronchial muscles, set up by breathing a cold atmosphere. In febrile cases, or where there is a tendency for the patient's temperature to remain high, Quinine should be given, or if badly tolerated Arsenic may be substituted, combined with Nux Vomica, Ammonia, and Cinchona. In aged patients with emphysematous lungs and a dilated right heart, it is often difficult to promote expectoration and maintain the heart's action. "In such cases we must give stimulants freely—a tablespoonful of brandy or whisky with a little hot milk and seltzer or Apollinaris water every hour; or in some cases champagne may be given if the patient prefer it," and a mixture of Ether Ammonia and Digitalis may be of use to stimulate the failing cardiac power, as Tr. Digit. ʒx, Sp. Ætheris ʒss, Ammon. Carb. gr. v, Aq. ad ʒi every 2 or 3 hrs. while required.

When convalescence is fully established, change of air to a bracing climate is beneficial, and will help to prevent recurrence or any tendency to the bronchitis becoming chronic. Diet may be ordinary but nutritious; and a little sound wine with meals may be allowed. A tonic, such as the following, is useful. R Tr. Nuc. Vom. ʒij, Ac. Nitro-hydrochl. dil. ʒij, Tr. Cinchonæ Co. ʒvj, Sp. Chlorof. ʒvi, Tr. Aurant. ʒvi, Infus. Calumbæ ad ʒvj, of which one tablespoonful may be taken thrice daily after meals.

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HÆMORRHAGIC DISCHARGES FROM THE VAGINA AFTER THE MENOPAUSE: THEIR SIGNIFICANCE AND TREATMENT.*

By WALTER TATE, M.D.,

Obstetric Physician to St. Thomas's Hospital.

THE subject which I have selected for our consideration to-night is one of much importance, and far too extensive to discuss in any detail. All that I shall attempt to do is to indicate the significance of certain signs and symptoms, and to suggest the general lines of treatment in doubtful cases.

First of all one must say a few words about the menopause. The menopause is the dangerous period in a woman's life for two reasons. Firstly, because a woman is always ready to attribute any irregular or excessive hæmorrhage occurring about this period to the "change of life." Secondly, because the average period at which the menopause occurs is also a very common time for the development of malignant disease of the uterus. In different patients the menopause presents varying features. In some women the periods get gradually less and less in amount, and finally cease. In others the flow may continue for longer than normal, then there may be a longer interval followed by another prolonged loss, and eventually the hæmorrhages get gradually less, and the intervals longer, till complete cessation occurs. In other women, again, the periods remain perfectly regular up to a certain time, and then suddenly cease. This cessation may even occur after a confinement or miscarriage.

In spite of all these variations in the symptoms and character of the menopause, it must be remembered that there is still a certain periodicity in the flow, and that sudden, severe, and prolonged hæmorrhages causing great prostration are not to

* Delivered before the Wimbledon Medical Society, May, 1901.

be regarded as *normal* menopause, but are cases which require careful investigation.

There is an idea among some women that there may be a secondary "change of life," and they will therefore regard a hæmorrhagic discharge as of little moment until its persistence and quantity causes them to become alarmed. Fortunately this belief is not very general, and, as a rule, where bleeding occurs after the menopause the patient recognises the fact that there is something wrong.

You will all, I am sure, agree with me in making it an absolute rule of practice that every case of irregular discharge which is blood-stained demands prompt local examination. It sometimes happens that medical men give way to the wishes of their patients in this respect, and postpone making any vaginal examination. Drugs are given, possibly ergot. Be the disease malignant or otherwise, the hæmorrhage is not unlikely to be checked for a time. The patient, of course, thinks she is better. Later on the hæmorrhage recurs; an examination is then insisted upon, and a diseased condition is found which might have been discovered before.

Treatment without examination is working in the dark. Sometimes no harm is done, but often weeks and months of valuable time are lost, and the patient's life is not only endangered, but actually sacrificed.

In considering the causes of post-climacteric hæmorrhage, we may for the purpose of this paper divide them into two groups. Firstly, those cases in which there is found, on examination, well marked and unmistakable evidence of disease; secondly, those cases in which the cause of hæmorrhage is obscure, the result of examination indefinite, and in which further investigation is needful to arrive at a diagnosis.

In the first group would be included :

1. Cancer of the vagina.
2. Cancer of the cervix, in which there is definite enlargement, with sprouting growth or marked ulceration.
3. Polypi projecting into the vagina, either adenomatous or fibroid.
4. Fibromyoma or malignant disease of the body of the uterus, in which the enlargement forms a definite pelvic or abdominal tumour.

In the second group one would include some of the more obscure or doubtful cases :

1. *Some* cases of cancer of the cervix.

2. Cases of uterine hæmorrhage where there is only slight or moderate enlargement of the body, and in which a certain diagnosis cannot be made by bimanual examination alone.

I do not propose to devote much attention to the first group of cases. Cases of cancer of the vagina, whether in the form of nodular infiltration or superficial ulceration, are readily detected; so also in the case of sprouting or ulcerated growths of the cervix, digital examination at once discloses the nature of the disease, and bleeding on examination confirms the diagnosis of malignancy.

As regards polypi, I shall have a few remarks to



FIG. 1.—Carcinoma of body of uterus, showing extensive infiltration of the muscular wall as far as peritoneal surface.

make later on. When they project into the vagina they can hardly be overlooked.

Finally, where there is marked enlargement of the uterus, giving rise to a definite pelvic tumour, it will be at once evident that there is gross disease of the uterus, and further steps will be taken to determine whether the enlargement is due to fibroid, sarcomatous, or carcinomatous disease. The following three cases are examples of marked enlargement of the uterus, in which the diagnosis was arrived at by means of digital exploration :

CASE 1.—Patient æt. 67. Menopause at forty-seven. Blood-stained discharge from vagina for three months before admission, with some abdo-

minimal pain. Some loss of flesh. Pelvic tumour, evidently uterine, reached to within two and a half inches of umbilicus. Cervical canal dilated under anæsthetic when tumour proved to be a sloughing fibroid.

CASE 2.—Patient æt. 49. Menopause occurred one year ago. There was a history of whitish discharge with irregular hæmorrhage for ten months before admission. The tumour formed by the enlarged uterus in this case reached within three inches of the umbilicus. Exploration of the uterus showed the case to be one of carcinoma of the body (see Fig. 1).

CASE 3.—Patient æt. 53. Menopause six years ago. There was a history of blood-stained dis-



FIG. 2.—Sarcoma of body of uterus.

charge for six months before admission. The uterus formed a tumour as large as a cricket ball. Exploration of the uterus proved the case to be malignant, and subsequent microscopic examination showed the disease to be sarcomatous (see Fig. 2).

I must pass on now to the second group of cases, viz. those in which examination of the patient does not disclose any very obvious disease. These are often the very cases in which it is so necessary to arrive at a definite opinion, and if we wait till the symptoms and physical signs are more pronounced we may find the temporising policy has seriously prejudiced the chances of recovery.

First of all, with regard to certain cases of cancer of the cervix, it must be remembered that

after the menopause the vaginal fornices frequently become obliterated, the vaginal cervix atrophies, and the whole organ undergoes diminution in size.

If malignant disease supervenes under these circumstances the enlargement, which is nearly always present in a malignant cervix, is easily overlooked. Remember, then, that carcinoma of the cervix may be present in a woman after the menopause without any very marked enlargement.

Moreover, where malignant disease occurs at this period of life it often begins deeply in the cervical glands, and slowly infiltrates the tissues of the cervix without causing any superficial ulceration. The cervix then may feel hard and enlarged, but does not at first seem to be the seat of serious disease. On closer investigation, however, it will be found that though the cervix is hard it is friable, and readily tears if grasped by a volsellum. One sign will always be present, namely, hæmorrhage on examination. The four features which should arouse one's suspicions in a doubtful case are (1) increase in size; (2) hardness; (3) friability; and (4) hæmorrhage on examination.

If doubt still exist in spite of the most thorough investigation, a piece of cervix should be removed, and a microscopic report obtained.

The next series of doubtful cases are those in which we find the cervix perfectly healthy, but in which there is slight enlargement of the body of the uterus. There is what one frequently refers to as a "bulky" uterus. Here it will probably be impossible to make a definite diagnosis by means of the ordinary bimanual examination. The history of blood-stained discharge suggests one of the following conditions:

1. Malignant disease of the body of uterus.
2. Fibroid polyp or submucous fibroid.
3. Adenomatous growth of endometrium.
4. Senile endometritis.

Slight enlargement of the uterus is consistent with all these conditions. The contour of the uterus does not enable one to distinguish between them. It used to be taught that the sound was of much help in the diagnosis. Fortunately, at the present the sound is much less used than formerly. Although it may be possible to detect irregularities of the inner surface of the uterus by means of its use, the information thus obtained is not sufficiently definite to warrant one in arriving at a decision between exploring the uterus and re-

moving that organ. Hæmorrhage, on passing the sound may occur in malignant disease, and in adenomatous growth of the endometrium; hence it is of no value in distinguishing between these two conditions.

In considering the four conditions just referred to one has to think of probabilities. Senile endometritis is a somewhat rare disease, in which the leading symptom is purulent discharge. The amount of bleeding is either very slight, or it may be entirely absent. Then, again, fibroid polypi are exceedingly uncommon at this period of life. Out of fifty-five cases of fibroid polypi which were treated at St. Thomas's Hospital in the ten years 1891—1900, only two cases occurred after the menopause. One of these was in a patient *æt.* 51, who had had the menopause at forty-nine. She had suffered from irregular hæmorrhage for six months. The other case occurred in a patient *æt.* 63, who had the menopause at forty-eight. The patient had had floodings for two months.

It is interesting to note that of the remaining fifty-three cases of polyp seventeen cases occurred between the ages of thirty and forty, thirty-five between the ages of forty and fifty, and one case occurred in a patient *æt.* 28.

If we put on one side senile endometritis and fibroid polypi as being rare causes of post-climacteric hæmorrhage, we are left with malignant disease of the body of uterus and adenomatous growth of the endometrium as the two likely causes.

There is only one point in the history which may help to distinguish these two diseases, viz. the presence of pain. If the patient has severe attacks of uterine pain (deeply situated in the hypogastric region) the disease is probably malignant. The absence of pain, however, does not prove the case to be simple, as it is present in only a certain proportion of the cases of malignant disease of the body.

In the majority of cases the only means we have of differentiating between these two diseases is by exploration of the interior of the uterus. To do this it is usually best to introduce a tupelo tent into the cervical canal, and to complete the dilatation under anæsthesia the following day. If the disease be simple adenomatous growth, one finds a variable amount of soft papillary growth—usually localised round the fundus and near the orifices of the Fallopian tubes. There is no

evidence of ulceration, or of any infiltration or hardness of the uterine wall around the growth. After the vegetations have been removed with the curette the inner surface of the uterus is quite smooth, and has the usual resistant feel when the curette is applied. In cases of malignant disease one may find masses of sprouting growth or areas of ulceration, more or less extensive, with hardness, due to infiltration of the uterine wall by growth (see Fig. 3). Between these two types, however, there are a certain number of cases in which digital exploration may give doubtful results. In all these a portion of the growth must be removed with the curette, and the pathologist must decide whether the disease is simple or malignant.

Adenomatous growth of the endometrium in



FIG. 3.—Carcinoma of body of uterus, showing extensive ulceration of the whole cavity with moderate invasion of muscular wall.

patients past the menopause must always be looked upon as a disease of doubtful malignancy. Some patients in whom this disease is discovered are entirely cured by one curetting. In others the hæmorrhage recurs after an interval, and the growth, though still microscopically innocent, is found to have recurred. In yet others the growths, after recurring one or more times, become definitely malignant. An example illustrating this is seen in the following case:

Mrs. W—, *æt.* 53, had the menopause at forty-eight. In January, 1895, in consequence of irregular hæmorrhage, which had continued for six months, the patient was curetted by Dr. Cullingworth. The scrapings were examined microscopically, and proved to be simple adenomatous

growth. She remained free from any discharge for two years and ten months, when watery discharge, at times blood-stained, again began. In April, 1898, the uterus was explored, and a large amount of soft growth was removed from the upper part of uterus. This was examined by Mr. Shattock, and proved to be columnar-celled carcinoma. She did not wish to have any further operation, as all bleeding and discharge had ceased, and decided to wait. After four months the watery discharge returned, and two months later the uterus was removed by Dr. Tate by the vaginal route. The uterus removed contained a large sprouting mass of rapidly growing carcinoma. It might be suggested that the disease in this case was malignant from the first. If this were so, it seems impossible to believe, and against all one knows of the course of malignant growths, that the patient would have remained perfectly well for nearly three years after simply curetting the uterus.

This is only one example of several which I have seen of simple growth becoming malignant. So much is one impressed with this possibility that I have advised removal of the uterus in patients past the menopause in cases where hæmorrhage recurs after a short interval, following exploration and curetting of the uterus for adenomatous growth of the endometrium.

I mentioned above that during the last ten years there had only been two cases of fibroid polypi in patients past the menopause. During the last eight years I have had under my care in St. Thomas's Hospital sixty-three cases in which the uterus was removed for malignant disease of the uterus. Sixteen of these were malignant disease of the body, and forty-seven were cases of malignant disease of cervix.

Fifteen of the cases of malignant disease of body occurred in women after the menopause, and no fewer than twenty of the cases of disease of the cervix, making thirty-five cases in all. If one added to these the number of cases in which the disease is too far advanced to admit of surgical interference, the frequency of malignant tumours of the uterus at this period of life would be still more striking.

It is necessary to say a few words with regard to mucous polypi of the cervix combined with malignant disease of the cervix or body of the uterus. It sometimes happens that a patient gives a history

of several months irregular bleeding, and on examination a mucous polyp is found projecting from the cervix. I have seen two cases in which the mucous polyp was discovered, and thought to be sufficient to account for the symptoms. Now, as a rule, mucous polypi growing from the cervix cause discharge, mucoid or muco-purulent, but little, if any, bleeding. Therefore one should



Site of mucous polyp.

FIG. 4.—Carcinoma of body of uterus with calcareous subperitoneal fibroid. (A small mucous polyp occupied lower part of cervical canal and projected into vagina.)

always make a very careful investigation in these cases, and not be satisfied with finding the mucous polyp alone. In one of the two cases, after removal of the polyp, the cervix was found infiltrated with carcinoma; and in the other, after exploration of the uterus, carcinoma of the body was found, with a calcareous subperitoneal fibroid (see Fig. 4).

The following propositions may, in conclusion, be made:

1. All cases of hæmorrhage occurring after the menopause demand prompt local examination.
2. When the cervix is found free from disease, digital exploration of the interior of the uterus should be advised at once.
3. The probabilities are strongly in favour of the disease being malignant.

It is only by careful attention to these three points that early diagnosis in cases of malignant disease can be made, and that we can hope to improve the results at present obtained by surgical interference.

A CLINICAL LECTURE ON MYASTHENIA GRAVIS.

Delivered at the National Hospital for the Paralysed and
Epileptic, Queen Square, W.

By F. E. BATTEN, M.A., M.D.

GENTLEMEN,—The condition which I wish to bring under your notice to-day is one that has attracted very considerable attention during the past few years.

The history of this disease (myasthenia gravis) is of some interest, for the first case was published by Dr. S. Wilks in the Guy's Hospital Reports in 1877, and he described it as a case of bulbar paralysis, in which no pathological changes could be found after death. In the following year Erb described a peculiar form of bulbar disease, the symptoms of which were ptosis, weakness of the jaw and neck muscles, weakness of the tongue and extremities, difficulty in swallowing, and weakness of the upper part of the face.

In 1887 two further cases were recorded, but it was not till 1891 that the disease was fully recognised, and the important papers of Jolly, Hoppe, and Goldflam were published.

Since that time numerous cases have been recorded and several digests written, of which that by Campbell and Bramwell published in *Brain*, 1900, is most valuable and complete.

The disease has been known under various names; the earlier cases were described as "cases of bulbar paralysis without any anatomical lesion." Asthenic bulbar palsy was suggested by Strümpell, but as Campbell and Bramwell point out, the affec-

tion is not limited to the bulbar muscles, and hence the name myasthenia gravis pseudo-paralytica is now most generally adopted, or, for short, myasthenia gravis.

Having thus given you a short abstract of the history of the disease, I propose to show you a case which was admitted to the hospital under my care. Before, however, bringing the patient in, I would give you a short history of the case from the notes taken by the house physician, Dr. Stanley Barnes.

M. F—, a girl æt. 14, complained of general weakness. The weakness was first noticed eighteen months previously. Her father died of cancer, and her mother suffers with the same disease. No history of any similar condition to that from which the patient suffers could be obtained, nor was there any evidence that any member of the family had suffered from insanity or epilepsy.

Up till the onset of this illness she was always healthy and quite like other children. She had, however, never been very strong, and had of late years grown very rapidly. The catamenia began recently, but her illness commenced some time before the first menstruation.

The history of the present illness is as follows:—About two years ago she was out in a thunderstorm, and got wet through. She caught cold, but was not laid up. Since that time she has steadily become weaker. The first weakness manifested itself by her falling down when walking. For quite a year, and probably for longer, she has had to throw her head back in order to see, because the eyelids drooped; she has never had double vision. Some difficulty in chewing and swallowing food was present among the earliest symptoms, almost eighteen months ago, and this symptom was worse at night, and she would be unable to swallow; she would then go to sleep, and when she woke up she would be able to eat her supper. She has been unable to wash or dress herself for a long time. Her weakness is always greatest at the end of the day and after exertion, but is much less when waking up in the morning. She is worse after anything that worries her, but variation in diet seems to make no difference to her.

She thinks she is worse just before her menstrual period, and better afterwards. She has had no headache or other trouble, and she is not troubled with constipation. She is a very tall, thin girl of fourteen years of age, her height being 5 ft. 9 in.

She is pale, and the chest is poorly developed. When sitting up there is a slight but distinct lateral curvature, with the convexity to the right, extending from the seventh to the twelfth dorsal vertebrae, and there is a well-marked compensatory curve in the lumbar region. The face is very expressionless, except immediately around the eyes and around the mouth. There is well-marked double ptosis, so that she does not look at the face of the interlocutor unless especially asked to do so, in which case she elevates the whole head and overcomes the ptosis for a moment.

There are no lines on the face. On attempting to smile only a small amount of movement immediately around the mouth come out, and the smile is very expressionless. She has practically no power either of elevating or corrugating her eyebrows. She can show her teeth feebly, and can whistle and blow out the cheeks. She is able to shut her eyes and mouth fairly well.

When asked to read aloud she starts off in a rather high, squeaky voice, which is clear and free from any nasal quality; as she progresses the tone of the voice alters and she seems to have difficulty in forcing sufficient air through the larynx to produce the necessary sound, and after a few minutes the voice dies away and she is no longer able to proceed, the difficulty appearing to be a respiratory one rather than any failure in the action of the laryngeal muscles, though at the same time the voice tends to assume a nasal quality.

All the special senses, sight, smell, hearing, and taste are good, and the optic discs are normal.

There is well-marked double ptosis, the upper and lower lid being separated by not more than about 5 mm. In the early morning the ptosis is less marked, but late in the day it becomes more marked. She can momentarily completely overcome the ptosis if asked to look up, but at such times the eyes do not follow the lids more than about 1 mm., which represents her maximum range of upward movement, and about 1 to 2 mm. represents the range of downward movement. Laterally the eyes move somewhat better, about 3 mm. outward and 1.5 mm. inward in each eye, but all movements are slow and do not look bilateral, though she says she does not have double vision. The pupils are equal, and react well to light and to accommodation. One cannot obtain any exhaustion of the sphincter iris by the action of light.

All the masticatory muscles are feeble and easily tired, and in order to assist these muscles she places the hand below the jaw when eating. She swallows fairly well when not tired, and fluids rather better than solids. The palate moves equally and well, and is not easily exhausted. The tongue can be protruded and put into either cheek, but its action is feeble, and it is somewhat atrophied. The mylohyoid and infrahyoid muscles are also feeble and easily tired. The muscles of the neck are weak, but all movements can be performed. The diaphragm and intercostal muscles act fairly, as do also the abdominal muscles.

Of the shoulder and arm muscles the serratus magnus is certainly weak, as shown by the winging of the scapula which takes place when the arm is thrust forward. The deltoid is very feeble and easily tired out, and the patient cannot hold her arm out at right angles for more than thirty seconds. The pectoralis major, the latissimus dorsi, biceps, and triceps are all feeble, but can perform all movements. The extensor and flexor of the wrist and fingers are feeble, but to a lesser degree than the upper arm muscles. The thenar and interossei muscles are fairly good; thus the proximal muscles are more affected than those more distally situated. The condition of the arms is symmetrical. With regard to the legs, the psoas and iliacus are feeble, the glutei and adductors are moderately so, the extensor and flexor of the knee are fairly good, and the extensor and flexor of the foot are good. In the lower limbs, again, the proximal muscles are more affected than those more distally situated.

Electrical examination.—The myasthenic reaction, *i.e.* the loss of contraction to a continued faradic stimulus is marked in the deltoid after half a minute; with the biceps it takes two minutes to obtain exhaustion, while in the flexors of the wrist it takes four minutes, and the reaction is not present in the thenar and interossei muscles. The muscles react readily to galvanism. The myotatic irritability is increased in all the muscles. The muscles generally are small and ill-developed, but there is no localised wasting.

Reflexes.—The palate reflex is diminished; the pupils react readily to light; the plantar reflex is flexor in type; the abdominal, epigastric, and jaw-jerks are not obtained.

The knee-jerks are equal and easily obtained,

and cannot be exhausted by repeated tappings, *i.e.* forty taps failed to make any difference in the character of the knee-jerk.

No affection of sensation can be detected.

The heart-sounds are normal; the pulse varies from 80 to 100. Nothing abnormal can be detected in the lungs or abdominal organs, and the urine was natural.

The case which has just been described is a fairly typical example of the condition known as myasthenia gravis, although it differs in some details; for instance, the bulbar symptoms are less marked than the ocular symptoms, and in the majority of cases the bulbar symptoms are in excess of the ocular.

The disease tends to affect the sexes alike, but it usually attacks women at an earlier age than men, for the former the age being about twenty, and for the latter about thirty-five. The disease is not hereditary, and does not seem especially to occur in neuropathic families. The onset of the disease is often ascribed to some illness, excitement, over-exertion, or to a cold, as it was in the present case, where the disease was attributed to getting wet in a thunderstorm. The actual onset of the disease is gradual and slight. Symptoms were present in the just recorded case for several months before they became marked. Ptosis is a very frequent and early manifestation, and with the diplopia, altered speech, and difficulty in swallowing and mastication, are among the earliest manifestations; the weakness of the limbs usually manifests itself later. The ptosis is sometimes constant, at other times only marked after the patient is tired. Local exhaustion of the levator palpebrarum has been produced by continued looking upward. The method by which these patients overcome the difficulty of looking up is by throwing the head back, as owing to the weakness of the frontalis the patient cannot overcome the ptosis by the over-action of this muscle, a condition which is frequently seen in cases of tabes with ptosis.

Weakness of the external muscle of the eyes is often present, and in some cases there is persistent ophthalmoplegia externa. The case that has just been shown is a good example of this.

One of the most constant symptoms of the disease is the difficulty in mastication; the difficulty may not be marked when the patient first begins to chew, but in a very short time the exhaustion of

the muscle becomes emphatic, and the patient is either absolutely unable to close the jaw without the use of the hand, or else it is so weak that a finger placed between the teeth of the patient cannot be bitten.

The weakness of the facial muscles is another very characteristic feature of the disease, and gives rise to the almost expressionless face, which is also well shown in the present case, the upper part of the face being more affected than the lower. The similarity between the face in myasthenia gravis and that which occurs in the facio-scapulo-humeral type of myopathy is well worthy of attention. The difference lies in the fact that in the Landouzy-Dejerine type there is no ptosis, no affection of the ocular muscles, no affection of the muscles of the tongue or masseter, nor is there any difficulty with articulation or deglutition, whereas in myasthenia gravis all these muscles may exhibit weakness in addition to those of the face.

Weakness of the palate is an almost constant symptom, and gives rise to the nasal voice, and to the regurgitation of fluid through the nose. The exhaustion of the palate is easily produced by making the patient repeat the word "Ah," when it will be noticed that the palate moves less and less, and finally does not move at all. As the patient reads aloud exactly the same change is noticed, for the voice becomes more and more nasal, and the words which at first were easily understood now become indistinct, the voice becomes weaker, the patient stops, seemingly unable to proceed owing to her inability to expel sufficient air from the chest for phonation.

The patient is generally unable to walk any distance; two or three times across the room is sufficient to give the patient the appearance of extreme exhaustion, and she walks as if she would fall to the ground. At times the legs may give way suddenly.

The disease usually affects the proximal muscles more than the distal, both in the upper and lower extremities, and that is so in the present case, since the myasthenic reaction is readily obtained in the deltoid muscles and not in the small muscles of the hand.

The muscles can be readily exhausted by repeated voluntary movement, and especially if the muscle be made to contract against a resistance. It is said that by exhausting one group of muscles

fatigue is caused in others, but this is by no means to be found in all cases, and is not present in the case shown this afternoon.

The muscles give the myasthenic reaction.

The myasthenic reaction is shown by applying the faradic current to a muscle and causing an active and continuous contraction. When first applied there is a brisk contraction, but this gradually becomes feebler, and finally ceases, and the muscle fails to respond to the strength of current originally applied, although by using a stronger current the muscle can again be made to contract.

If after exhausting the muscle a rest be given, and the same strength of current be applied a contraction is again obtained, which, as the current is continued, again becomes less and less.

If a muscle which has been completely exhausted to faradic stimulation be stimulated by the galvanic current the muscle responds normally, and even after repeated galvanism no exhaustion of the muscle to that current can be produced.

Atrophy of muscles is an unusual manifestation in this disease, in fact, the presence of such atrophy would lead one to suspect that the case was not one of myasthenia; on the other hand, cases which must be classed as myasthenia gravis have been reported in which muscular atrophy has been present, and even in the case shown this afternoon the tongue is so thin that its appearance suggests that atrophy has taken place. Fibrillary tremor is not, as a rule, present, and certainly cannot be considered as an ordinary manifestation of the disease.

With regard to the reflexes, the superficial and deep reflexes are usually present, and show no alteration in character. It has, however, been noted in certain cases that the knee-jerk can be abolished by repeated tapping. There is never any affection of sensation, and the sphincters act normally.

Course.—The course which the disease runs is usually progressive, but in some of the cases on record recovery has taken place.

The following description, taken from a case that was recorded by Dr. Morley Fletcher and myself, gives a good idea of what is often the final stage of the disease.

A girl, *æt.* 22, gave a typical history of the disease, and had the usual difficulty in swallowing. To repeat the various details of the case is un-

necessary, but the girl at first seemed to make some improvement. One day when she was taking her dinner, and, as usual, was being fed by the nurse with milk and fish, she suddenly said she could not swallow, and said, "I am choking." She became very blue, and respiration ceased; there was no struggling, no recession of the chest, but merely an absence of respiratory movement. Artificial respiration and oxygen were resorted to, and the cyanosis became less; strychnine was given—some respiratory movement now took place, but it was limited to the sterno-mastoid and accessory muscles of respiration. During the whole of this time the pulse showed no evidence of failure, remaining slow and of good volume. The patient was perfectly conscious, raised her hand to her mouth, as if to aid her respiration by elevating her jaw, but the efforts at respiration became feebler, the action of the heart became more rapid, and the patient died. Tracheotomy was performed in this patient shortly before death, in the hope that that might allow a freer entrance of air into the chest, but it is obvious that the failure was in the respiratory muscles, and was not due to any obstruction in the larynx.

A girl who was in the hospital a short time ago died in almost exactly the same manner, and in that case also tracheotomy was performed. Another case of sudden death is recorded by Prof. Clifford Allbutt in the article on this affection in his 'System of Medicine.'

Diagnosis is, as a rule, not difficult, the history of increasing weakness, the rapid fatigue of the muscles after exertion, the involvement of the upper part of the face and the ptosis are a group of symptoms which make the disease easy of recognition. In addition to the above, if further evidence be needed, is the distinctive reaction of the muscles on faradic stimulation.

The facies of these patients often resemble that type of myopathy known as the facio-scapulo-humeral; the diagnosis between the two conditions is not, however, difficult, as the absence of any affection of the ocular muscles, or of the tongue or palate in that type of myopathy, together with the history, are usually sufficient to make the diagnosis clear apart from the atrophy of the muscles of the trunk in these cases.

Some cases have been mistaken for hysteria, and the variation in symptoms and the difficulty in

swallowing which these patients manifest certainly suggests such a diagnosis; on the other hand, the weakness of the palate, the ptosis and the affection of the masseters should make the diagnosis clear.

From the other forms of bulbar palsy myasthenia is distinguished by the variability of its symptoms and the affection of the ocular muscles.

The difficulty of distinguishing the disease from an acute polio-encephalitis—an affection of the cells of the cranial nuclei—is one which is great, and in some cases would seem impossible. The history of sudden onset and the absence of any affection of the muscles of the trunk would be points in favour of the diagnosis polio encephalitis, rather than myasthenia.

Morbid anatomy.—The original name, as applied to this disease, viz. "bulbar paralysis without anatomical change," indicates what is known of the morbid anatomy. Although this was the condition described by observers many years ago, almost all pathologists with the most recent methods have failed to find any anatomical change in the nervous system. Some few months ago, however, Laquer described a case which exhibited symptoms which were regarded as diagnostic of myasthenia gravis. The patient died, and on pathological examination by Weigert* a new growth was found in the thymus gland, and also secondary growths in the muscles. It seems altogether unlikely that had similar changes been present in the muscles of other cases of the disease that they should have escaped observation. This observation, however, of Weigert's would suggest that in future examination of the condition of the muscles will have to be most carefully investigated.

I have had the advantage of examining two cases of this disease. In the first case, published with Dr. Morley Fletcher, certain changes were present in the cells of the third, seventh, and ninth nuclei, *i. e.* there was alteration in the shape of the cell, blunting of the processes, and alteration in the character of the chromophilic substance. In another case changes of a similar nature were shown to be present in some of the cells of the third, sixth, and twelfth muscles, but side by side with these altered cells were cells which were perfectly normal. (The nature of these changes

was shown by means of the lantern at the end of the lecture.)

Pathology.—The true pathology of this disease is as little known as the morbid anatomy.

The symptoms and course of the disease suggest that it is due to some altered metabolism or toxin. What the nature of this toxin is, whether of endogenous or exogenous origin, whether dependent on the internal secretion of some organ, or on the failure to excrete certain waste products has yet to be proved.

Campbell and Bramwell express the opinion that the disease is of microbic origin, and in support of this theory point to the fact that the disease follows on such maladies as influenza, scarlatina, typhoid, etc. The next question which arises is, On what structure does the poison act? Is it an affection of the upper neurons, the lower neurons, and muscle-end plates, or of the muscle fibres themselves?

The peculiar myasthenic reaction of the muscles, the atrophy which they occasionally undergo, would certainly seem to point to a lesion of the lower motor neuron. The fact that after the muscle has been completely exhausted to faradism it will still react to galvanism would seem to indicate that the affection is one of the lower motor neurons, and not of the muscle itself, since the faradic current acts chiefly on the nerve, while the galvanic current acts chiefly on the muscle.

On the other hand, it should be borne in mind that even after a muscle has been completely exhausted by faradism it will still contract strongly to voluntary impulses, and therefore the lower motor neuron must be still capable of transmitting impulses. The only point in favour of regarding the condition as a muscular one is the pathological change found in the muscle by Weigert. The balance of evidence, however, is in favour of the affection being one of the lower neuron, but whether of the cell-body, the neuraxon, or the motor nerve ending cannot be determined.

Treatment.—With regard to treatment, massage and faradism seem to do actual harm, exhausting the patient, and giving rise to a great feeling of weakness. Complete rest in bed is useful for a time, and considerable improvement often takes place. The difficulty which the patient has in mastication makes it essential that the food be soft. Even when the difficulty in swallowing

* 'Neurologisches Centralblatt,' 1901, p. 597.

becomes very great it is inadvisable to pass an œsophageal tube, and it is best to allow the patient to rest for a while, and then attempt to feed her again.

With regard to drugs, strychnine has done some good; thyroid, thymus, and supra-renal extract have all been tried, but with little definite success. For the present case the patient has certainly improved while under suprarenal extract, which was given at the suggestion of Dr. Hughlings Jackson, who pointed out that the position of the eyelids was the exact reverse of that produced by the action of suprarenal extract.

Increase in the amount of fluid given to the patient made but little difference; diminution of the amount certainly made the patient worse.

MYASTHENIA GASTRICA: ITS DIAGNOSIS AND TREATMENT.

By GEORGE HERSCHELL, M.D.Lond.

MUSCULAR weakness of the stomach, or gastric myasthenia, although usually unrecognised by the practitioner, is undoubtedly one of the commonest causes of dyspepsia. When it was first found possible to extract the contents of the stomach and to test the quantity and composition of the gastric secretion, it was thought that we had at last obtained a key to the diagnosis and treatment of the different disorders of digestion. Later experience has shown this hope to be fallacious, and it has now been established beyond the possibility of contradiction that abnormalities in the motor power of the stomach play a far more important rôle in the production of those affections which are accompanied by the collection of symptoms which for convenience we term "dyspepsia."

If the motility of the stomach be normal, and if it is able to empty itself at the proper time into the duodenum, alterations in the composition of the gastric juice will usually produce no symptoms whatever. The stomach, of course, not being ulcerated, inflamed, nor abnormally sensitive and hyperchlorhydria being excluded. Thus we may have gastric juice absolutely innocent of HCl or pepsin, and no symptoms will be produced as long as the stomach can discharge the food into the duodenum before fermentation has taken

place. Conversely, no matter how normal the gastric juice, if the stomach be unable to completely empty itself before the breakfast of the following day, stagnation and fermentation of the retained food residues will take place, and the patient will suffer severely.

This fact—that the work of the stomach is mainly that of preparing the food for more complete digestion in the intestine—was pointed out as far back as 1894 by Fleiner* and Bourgetans,† and is completely established by instances of the restoration to practically perfect health of subjects who have undergone complete excision of the stomach. There is really far greater analogy between the stomach of a man and the gizzard of a bird than would appear at first sight. Both organs are muscular, and both triturate and disintegrate the food with the help of certain secretions, and, when properly prepared, pass it onwards into the next portion of the alimentary canal. The older views, as to the dependence of the symptoms of the so-called atonic dyspepsia, mainly upon deficiency of acid and pepsin in the gastric juice, may therefore be taken to be quite obsolete. Such being the case, it behoves us as rational practitioners of medicine to revise our methods; and if we would really relieve our cases of dyspepsia, devote very serious study to muscular weakness of the stomach wall. In this short paper myasthenia gastrica will be briefly discussed with special reference to diagnosis and treatment.

Gastric myasthenia is essentially a local weakness of the muscular wall of the stomach. A stomach thus affected cannot support the weight of a normal meal without stretching, and is unable to contract efficiently upon its contents. As regards its ætiology:

1. IT MAY BE CONGENITAL.—Some unfortunate individuals are born with an abnormally small amount of muscular tissue in the walls of the gastrointestinal canal, and suffer from constipation and indigestion from early childhood.

2. IT MAY BE ACQUIRED.—Habitual over-distension of the stomach by food and drink may produce it. If you pump fluid into a rubber ball, you will find that after a certain degree of distension it will absolutely lose the power of contracting to its

* 'Sammlung klin. Vorträge,' No. 103, 1894.

† 'Wien. med. Blätter,' No. 45, 1894.

original size; and the stomach behaves in very much the same manner to long-continued ill-treatment. It is thus very common among habitual excessive drinkers of beer and tea, and among those who eat in haste without proper mastication. Constant use of hot beverages appears to be especially liable to weaken the muscular walls of the stomach, and it is thus probable that it is not the tannin in tea nor yet the alkaloids which produce dyspepsia, but simply the mechanical weight of the hot liquid. I have met with many cases of myasthenia of the stomach among patients who have tried to cure themselves of dyspepsia by means of the so-called Salisbury treatment.

3. IT MAY OCCUR PATHOLOGICALLY FROM—
 (a) *Defect in the nutrition of the Stomach Wall.*—In this connection it is most commonly met with in states of general malnutrition, such as in convalescence from acute diseases. In such cases it may be directly induced by the incautious administration of a large meal. From this we learn the important lesson that we are acting wisely in restraining the appetite of the patient, and in only allowing him to resume the ordinary mixed diet of health by very gradual steps. In wasting diseases, such as phthisis, the stomach will participate in the general tissue starvation, and the disease may be induced unless great care is taken that the patient's meals shall be in a finely divided condition, and not in excessive quantity at any one meal. (b) *Disease of the Stomach Wall.*—Myasthenia is not unfrequently met with as a complication in cancer, but its commonest cause under this heading is chronic gastritis. In this case the weakness of the muscular substance of the stomach is produced much in the same way as that of the myocardium in pericarditis, the superficial layers of the muscle immediately underlying the gastric mucosa showing evidence of fatty and granular change. It also frequently follows the passive congestion of the stomach due to obstruction in the portal circulation. It may thus be one of the first signs of cardiac failure. In some cases it is apparently due to irregular gout manifesting itself in the stomach. (c) *Defective Innervation of the Stomach.*—Mild degrees of myasthenia are frequently met with as a local manifestation of general neurasthenia. In fact a large proportion of all cases of neurasthenia manifest themselves at one time during their course as gastric atony.

We may divide cases of myasthenia into three degrees according to severity.

First degree.—The stomach empties itself into the duodenum later than normally, but succeeds in doing so completely before the next meal. In the very early stages digestion is prolonged, but there may be no symptoms. But sooner or later a time comes when the stomach is hardly empty when the next meal-time arrives. We thus approach the second degree.

Second degree.—The stomach cannot empty itself before the next meal, but does so during the night. The stomach is thus able to take a short rest before breakfast. This degree has been termed by Van Valsah and Nisbet* the "stagnation form," because the food has time to stagnate and ferment before it is finally expelled from the stomach.

Third degree.—The stomach can never completely empty itself, but always contains food residues from previous meals. This has been termed the "retention form" by Van Valsah and Nisbet, and corresponds to the atonic dilatation of other writers.

Of course the affection does not pass abruptly from one stage to another. There is, for instance, usually a transitional period between the second and third degree, in which the stomach manages to completely empty itself occasionally.

In practice it is convenient to group together the first and second degrees, as in these there is no retention of food residues, and we thus have the classification of Van Valsah, Hemmeter, Riegel, and others, viz.:

(a) Myasthenia without retention, a comparatively mild disease, and

(b) Myasthenia with retention, an affection of extreme gravity.

It is clinically convenient to thus divide and study the cases, as although, of course, the groups are successive stages of the same affection, they differ materially in the treatment required and in prognosis.

Myasthenia without Retention.—In this group we have the first and second degrees of myasthenia. In the first, as we have already pointed out, the stomach empties itself slower than it should do, but yet with sufficient rapidity to

* 'Diseases of the Stomach,' Rebman, 1899, p. 350.

prevent fermentation from taking place. In the second degree, it only succeeds in evacuating its contents during the night, with the obvious result that stagnation of food and consequent fermentation may occur. The extent of this will depend mainly upon the fermentable nature of the food, and whether or not germs have been ingested with it from septic teeth and gums.* It is needless to say that fermentative changes never take place to anything like the same extent as in the third degree, where the stomach practically never empties itself without assistance, and there is consequently permanent retention of food residues.

Van Valsah and Nisbet (op. cit., 1899, p. 350) have drawn attention to the fact that myasthenia of the first degree may exist to a certain extent without the production of symptoms, and following them we may conveniently divide the evolution of the disease into two stages—a latent period, during which there are practically no symptoms, and a clinical period, when the affection is accompanied by the *syndrome* indigestion, and during which the patients come to consult us.

It would appear as if gastric myasthenia must reach a certain degree of intensity before symptoms of indigestion can be produced; this will account for the differences of opinion which have been expressed by competent observers as to the significance of the splashing sound which can be produced by appropriate manipulation of the abdominal walls over the region of the stomach. It has been asserted that this symptom was of little significance as a diagnostic sign of myasthenia gastrica, because in many cases it could be elicited in individuals who had apparently absolutely no digestive troubles. For the production of the splashing sound it is necessary that the stomach should contain a mixture of gas and air. Theoretically, therefore, it should never occur in a really healthy stomach, which should always be in a state of contraction upon its contents. It must be borne in mind that the stomach is not a simple bag of a definite size, but a muscular organ whose internal capacity, when absolutely normal, is exactly that of the bulk of its contents. If you introduce a pint of water into the healthy stomach, its interior measures exactly a cubic pint, and there is no room

for anything else. Any air which may be swallowed, or gas which may form in it, is immediately expelled. It follows, therefore, that theoretically no stomach in which splashing can be obtained can possibly be entirely healthy as to its musculature. Probably such a perfect stomach is very seldom met with at the present day, and we are therefore in the habit of saying that in a normal stomach splashing can usually be met with directly after a meal. We may, however, I think, assume that all splashing occurring later than one hour after a meal must indicate some degree of muscular weakness of the stomach, and patients in whom this physical sign is accidentally discovered should not neglect treatment, although they are not consciously suffering from any gastric trouble. In many cases the patient has become so accustomed to his slight indigestion that he does not notice it, and will not complain about it unless cross-examined upon his sensations after meals. Numbers of patients in whom I have discovered gastric splashing during the digestive period, and who have assured me that they were not troubled with indigestion, have admitted, on close questioning, that they were conscious of a slight feeling of fulness during digestion, or frequently brought up wind, or were troubled with constipation. In many cases symptoms such as headache, migraine, or agoraphobia, not suggesting dyspepsia to the patient, but having a distinct time relation to food, and therefore obviously of gastric origin, have been overlooked.

According to Van Valsah and Nisbet* the clinical period is apt to supervene suddenly, and is apparently induced by mental shock or overwork, or an excessive meal. The patient is thus apt to ascribe the onset of his complaint to a Masonic banquet or public dinner at which he exceeded the dictates of prudence. Patients who are in the latent period will often tell you that after such a large dinner they have an attack of indigestion during the night. The slightly myasthenic stomach, although still well able to handle an ordinary meal, is yet unfitted to deal effectively with one of unusual bulk. Such a patient will retire to bed feeling extremely comfortable. He has had the ordinary public dinner which many men can take several times a week with impunity.

* *Vide* the Author's paper "On Oral Sepsis as a Modifying Factor in Affections of the Stomach," 'International Medical Magazine,' June, 1901.

* Op. cit., p. 350.

He has had, perhaps, various assorted hors d'œuvres, soup, whitebait, another fish, two entrées, the inevitable saddle of mutton, bird, and salad and sweets. He has taken about half a bottle of champagne, a coffee, a liqueur, and afterwards a whiskey and soda, and has smoked a couple of cigars. He considers that he has been most moderate because he has taken no iced pudding, no fruit, and has not mixed his wines. He retires to bed and falls off to sleep. Now, a normal stomach would, even if unable to completely digest such a meal, empty itself in a few hours into the duodenum, and thus shift the onus of disposing of it upon the intestine, perhaps with the result of a little diarrhoea. The myasthenic stomach, however, cannot cope with it as it is too heavily handicapped. The patient will shortly wake with nausea and a sense of discomfort in the region of the stomach. In many cases the temperature is slightly raised, and there may be trembling or jactitation of the limbs, or a feeling of soreness or chilliness over the body. In some cases I have seen a distinct slight rigor. Usually vomiting speedily occurs, and the patient drops off to sleep much relieved.

SYMPTOMS OF THE CLINICAL PERIOD.—1. *First degree*.—The symptoms of this degree are not distinctive, as there is no fermentation in the stomach. We may meet with—

(a) Fulness after meals experienced in the region of the stomach. One must be careful not to confound this with the sensation of fulness in the lower abdomen so common in intestinal indigestion of starch. Patients have a very vague idea as to the position of the stomach, and should always be asked to point to the exact spot where they feel the pain.

(b) Eructations of gas, with or without slight regurgitation of food.

(c) Inability to sustain prolonged mental or bodily exertion. This symptom is, of course, the expression of a neurasthenic condition, but whether the result of the gastric myasthenia or a concomitant it is difficult to say.

2. *Second Degree*.—In addition to the symptoms enumerated above we shall have, depending upon the additional factor, the stagnation and fermentation of the food.

(d) Bitter or acid taste of the regurgitated matters.

(e) Symptoms probably due to the absorption of toxins from the stomach—

1. Anorexia, nausea, and vomiting.
2. Insomnia, headache, vertigo, mental depression, migraine.
3. Various paræsthesiæ, numbness, tingling, sensations of crawling.
4. Fibrillary twitchings and limited clonic contraction of muscles.
5. Slowness of the heart's action, with, perhaps, reduplication of the second sound at the apex.
6. Certain skin eruptions, notably erythema and urticaria.
7. Anæmia.

(f) Intestinal symptoms, of which the most important are—

1. Muco-membranous colitis.
2. Intestinal indigestion.

As long as the intestines remain normal, it is possible for the greater part of the digestive process to take place there, and the patient may retain his nitrogenous equilibrium. When this is no longer the case, the patient is within a measurable distance of progressive emaciation. It is obvious that in gastric myasthenia the intestines are exposed to special dangers from the large quantity of decomposing material which is continually being poured into them. As already stated, the commonest intestinal trouble met with in these cases is muco-membranous colitis, and in cases of this affection the presence of gastric myasthenia should always be looked for as a possible cause.

(g) In some cases the irritation of offensive food may set up a chronic glandular gastritis (aptly termed by Van Valsah and Nisbet "chronic hypersthenic gastritis or gastritis glandularis proliferans," op. cit. p. 414), and we may have the case complicated with the symptoms due to the excessive secretion of HCl. These are burning epigastric pain, acid risings, and, very often, persistent headache.

It will thus be seen that, although interesting, the symptoms are not entirely characteristic, and of themselves will not enable us to make a diagnosis. The following three points are, however, suggestive, and should lead us to suspect the condition of things:

1. The symptoms only occur when there is food in the stomach, and the distress *varies directly with*

the amount of food taken. When the stomach is empty, the patient feels perfectly well.

2. Liquids give rise to as much discomfort as solids, sometimes even more.

3. The patient is adequately nourished, and maintains his metabolic equilibrium. But all these symptoms offer merely presumptive evidence, and to establish upon a sure foundation the nature of the case we must proceed to make a physical examination.

PHYSICAL EXAMINATION.—First of all, we must disabuse our minds of the idea that the estimation of the actual size of the stomach will help us to any considerable extent. Stomachs may vary in size the same as other organs of the body, and a large one may very well be able to empty itself at the proper time, whilst an exceedingly small one may happen to be atonic and myasthenic. One finds in the text-books and monographs on diseases of the stomach many precise directions for making out the exact size of the stomach. In practice one finds that without the assistance of inflation or other intra-gastric means it is exceedingly difficult, and such being the case it is a fortunate circumstance that such estimation is of so little practical value. The sum total of our present knowledge may be summed up in the statement that if the stomach is manifestly large it is more likely to be myasthenic than if below the usual size. Beyond this we cannot go.

In the diagnosis of gastric myasthenia we rely upon the following signs :

1. *Splashing.*—As we have pointed out already, splashing should theoretically not be present in a normal stomach during the digestive period, and its presence should be strongly presumptive of myasthenia. We cannot do better than again quote Van Valsah and Nisbet : * “ This (splashing) alone is sufficient to establish the diagnosis of motor insufficiency when the proper precautions are taken.” Again, “ These splashing sounds are usually absent in the healthy vigorous stomach at all moments of the digestive period, except towards the end of the digestion of a very large meal. The myasthenic stomach splashes throughout the digestive period, and also later than the moment when the normal stomach should be empty, the evacuation of the stomach being delayed.” As the technique for the

production of the splashing sound will be found in the different monographs, and has been fully described by me elsewhere,* there is no necessity to go further into the subject in the present paper.

2. *The production of splashing when two glasses of water are taken by the patient upon an empty stomach [that is, in a stomach which did not splash until the water was taken].*—If the stomach is healthy most usually no splashing can be elicited after the ingestion of sixteen ounces or so of water, as the stomach will contract tightly round its contents. Sometimes, however, it can be obtained under these conditions, but only for a few moments, as the stimulus of the examination will quickly cause contraction of the stomach to take place. The myasthenic stomach, on the other hand, sags down under the weight of the contained water, as in Dehio's test (see next paragraph)—and does not completely empty itself for at least two hours, during which period splashing can be obtained. This peculiar inability of the myasthenic stomach to dispose of liquids is quite characteristic, and may be demonstrated by the water test to be presently described.

3. *Method of Penzoldt and Dehio.*—This method depends upon the fact that if a person whose stomach is healthy drinks a couple of tumblers or so of water, the position of the dulness corresponding to the greater curvature will remain practically unchanged. A myasthenic stomach, on the other hand, will give way and stretch under the weight of the contained liquid, and the lower limit of the stomach will vary accordingly.

To perform this test half a glass of water is first of all drunk by the patient, and the lower margin of the stomach is made out as accurately as possible by percussion whilst the patient is in the upright position. It is marked with a dermatographic pencil. The patient then drinks about a pint of water in doses of half a tumblerful at a time, the lower margin of the stomach being percussed out between each. If the stomach is myasthenic this will be found to be distinctly lower after each draught.

In a case of ordinary severity we may be reasonably satisfied with these three tests. In actual practice it is not always either necessary or advisable to subject the patient to the inconvenience of intra-gastric instruments. If the splashing is limited

* Op. cit., p. 353.

* ‘ Indigestion,’ 2nd edit., p. 106.

to the digestive period, and is absent before breakfast, we may assume myasthenia of the first or second degree. And in this, as in many other affections met with in daily practice, we may be content with such a reasonable certainty, and, unless the patient expressly wishes it, may abstain from the final absolute proof. In other words, in ordinary daily practice we shall be justified in diagnosing myasthenia of the stomach and treating the patient for it from the results of these three foregoing tests. But it is in our power in doubtful cases to absolutely demonstrate the existence of myasthenia beyond the possibility of error by the following methods:

4. *The water test.*—This is, I believe, the invention of Van Valsah and Nisbet, and is a very ingenious method of estimating how much water passes out of the stomach in a given time. The principle is very easy to understand. If you introduce, for example, 100 c.c. of water into the stomach, and some time afterwards another 100 c.c. containing 1 per cent. of sugar, and if you now extract some of the stomach contents and find that the percentage of sugar is still 1 per cent. it is obvious that the whole of the original 100 c.c. of water must have passed out of it. If you find the quantity of sugar to be $\frac{1}{2}$ per cent. then it necessarily follows that one quarter of the original quantity of water still remains in the stomach, *i.e.* 50 c.c., and so on. The test is actually performed in the following manner: 300 c.c. of water are given to the patient before breakfast. One and a half hours later he swallows 100 c.c. water containing 1 per cent. of sugar, and the stomach region is well kneaded, with the patient in the recumbent position, so as to well mix up the stomach contents. You now pass a stomach-tube and extract by aspiration or expression a few c.c. It does not matter how much. You now test this quantitatively for sugar by Pavy's method.

Then the formula

$$100 \div \text{per cent. of sugar} = 100$$

will give the number of c.c. of the original 300 c.c. of water remaining in the stomach. A normal stomach should have completely disposed of the original quantity of water by this time, and if any be found remaining then we are absolutely certain that myasthenia exists. This test offers us a most practical method of excluding the stagnation of stomach contents due to pyloric obstruction. In

all except the extreme degrees of pyloric stenosis the stomach, whilst more or less unable to deal with solids, is yet quite capable of emptying itself of liquid, as its muscular power is unimpaired. A myasthenic stomach, on the other hand, finds its greatest difficulty in dealing with water and thin fluids. Of course, we must not forget that the water test will fail in those rare cases of pyloric obstruction which are complicated with myasthenia. Such cases are not frequently met with, because naturally the effect of long-continued gradually increasing obstruction of the pyloric orifice of the stomach is to cause hypertrophy of the muscular layers of the stomach to take place.

5. *The result of expression or aspiration of the stomach contents as to quantity one hour after an Ewald test breakfast.*—In a normal stomach we should not be able to obtain more than 125 c.c., whilst a myasthenic stomach finds so much difficulty in emptying itself that we can usually extract at least 150 c.c., probably much more.

6. *The finding of food residues in a stomach six or seven hours after an ordinary mixed meal.*—By this time a normal stomach will have emptied itself. If, in addition to this, we find no food residues before breakfast, then we are certain that the myasthenia is only of the first or second degree. Of course, in the earlier stage of pyloric obstruction we may find the same food residues, but this condition may be readily differentiated by the water test already described. If we find food residues before breakfast the case is myasthenia of the third degree.

By these various methods it is one of the easiest things to establish the presence or absence of gastric myasthenia without the possibility of error, and there is therefore no longer any excuse for the treatment of a patient for "indigestion" without doing so.

(To be continued.)

MR. MARMADUKE SHEILD in the course of an address on the Indications for Operative Treatment in Appendicitis, delivered at the opening meeting of the Windsor and Eton Medical Society on October 23rd last, in referring to the question of purgation in peritonitis, said he had lately independently adopted a method which he believed would be of great utility. His earliest case was in 1899, but he had not until then mentioned publicly his practice of introducing purgatives into the cæcum at the time of operation. He usually introduces two or three drachms of sulphate of magnesia, with two drachms of glycerine, some nux vomica, and water through the stump hole of the appendix with an ordinary syringe. Three hours after, a turpentine enema is given, and the results have in five cases been most striking.

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A CLINICAL LECTURE

Delivered at the West London Hospital,

By F. SWINFORD EDWARDS, F.R.C.S.

GENTLEMEN,—Affections of the prostate may be divided into two groups, namely, those occurring before middle age, and those met with in the aged.

I suppose the pathological condition most frequently met with before middle age is prostatic catarrh. Next in order is acute prostatitis, and prostatic abscess—which is generally gonorrheal in origin, but may be traumatic.

Tubercular disease frequently attacks the prostate, and a gouty prostatitis is not unknown in the young adult. Prostatic calculi, or concretions, are rare, but have to be reckoned with.

The consideration of prostatic catarrh is really a continuation of our lecture of the other day, in which we discussed gleet. Prostatic catarrh, as a rule, is the outcome of antecedent gonorrhœa, and has to be treated in the way I have recently indicated. Now and then prostatic abscess follows, that is to say, a prostatitis lit up by gonorrhœa, results in abscess. Now, this abscess in the prostate gives rise to great pain in passing water, constant desire to micturate, with a sense of fulness and weight in perineum and about the rectum, and sometimes the passage swells so much as to cause retention, for which a catheter often has to be passed. This may relieve your patient in more ways than one, because you may not only draw off the water, but you may rupture the abscess, and the patient may, in this way, be relieved of the obstruction. But it does not always follow that abscess which has ruptured into the prostatic urethra gets cured very easily. Something more than leaving it to Nature may have to be undertaken. What would you do in such a case? You may try bladder irrigation such as I described on the last occasion, throwing in a pint of some antiseptic solution without the passage of a catheter, the patient then evacuating the contents of his bladder in the ordinary way

which washes out both the abscess cavity and, indeed, the entire lower urinary tract. But this does not always suffice. You may try what irrigation of the prostatic urethra will do by means of a long tube passed into the prostatic urethra, and to which a Higginson's syringe is fixed, or you may instil a solution of nitrate of silver. But what I have found very useful in these cases is massage of the prostate *per rectum*. I recollect it was first brought under my notice by some patient coming up to me and asking me if I thought so-and-so could be a decent, upright, and honest surgeon, because he practiced massage of the prostate, throwing, therefore, a doubt upon the man's respectability. I said I had not heard very much about it, but that it was done in Germany and America, and that it seemed to commend itself to me in some instances. I thought over the matter, and recollected certain cases I had come across where it might have been practiced with benefit. I have since done so more than once, both in prostatic catarrh, in chronic abscess of the prostate, and in abscess of the vesiculæ seminales, with very great benefit to my patients. Bearing on this point, I will read you notes of a case, which was the first case I had. Mr. O. was sent to see me in 1898 with this history:—He had had his second attack of gonorrhœa, which had lasted five weeks; but not getting any better, he sought further advice, and came to me, recommended by a friend. I sent him into a private home, and ordered him hot baths, with a suppository of iodide of potassium—a suppository which we will talk about later on. He got better under this treatment in a week, but during the time, an abscess broke into the urethra. After this he used a prostatic irrigator with a permanganate of potash solution, and he left, feeling almost well. I saw him again, not quite a month afterwards, and I have a note to this effect:—"Symptoms have all recurred. Examination per rectum shows prostate very large, hard, and tender on the right side." I sent him again into the nursing home for prostatic massage, which I practised daily. He went home in a fortnight, and my note says:—"Very much better. He is taking sandal-wood oil capsules, two of them, three times a day." I asked him to come to my house daily for massage. My note is, "Massage caused a flow of pus from the urethra." On September 9th the note is, "Is well; has now no discharge."

This is the history of several other cases which I have had since—cases which probably would not have got well without something of the kind. For its practice I recommend the genu-pectoral position. It need not take any more than two or three minutes. The finger, well coated with vaselin, is passed into the rectum until the prostate is felt, and then forcible stroking of the prostate should be done from above, trying to express downwards any discharge which may be in an abscess cavity in the prostate into the urethra, and so getting rid of it. In like manner you may practise massage of the vesiculæ seminales for chronic vesiculitis. An inflammation of the prostate may give rise to a periprostatis resulting in abscess, and that abscess may open into the rectum. I have known several instances of blind internal fistulæ as the outcome of gonorrheal prostatitis resulting in abscess. A periprostatis abscess generally occupies what is called the pelvi-rectal space, that is to say, it is above the levator ani muscle and outside the muscular coat of the bowel. You recollect that abscess in connection with the rectum may form in one of four spaces, under the mucous membrane, that is to say, internal to the muscular coat of the bowel, outside the muscular coat of the bowel, and internal to the levator ani, and usually results from prostatic abscess. Then there is the abscess which forms in the ischio-rectal fossa, also the marginal abscess which forms under the skin in the neighbourhood of the anus. Of these, the abscess which forms in the pelvi-rectal space is the most difficult to treat. But we will not go into that, as it rather comes under the head of rectal surgery. I might say that all prostatic abscesses should be treated from the perineum. As soon as you are sure that you have a prostatic abscess to deal with, which seems unlikely to rupture into the urethra, it is best to give an anæsthetic, put the patient into the lithotomy position, and make a dissection down through the perineum towards the apex of the prostate, somewhat as if you were going to do Cock's operation for the relief of retention of urine. When fluctuation is present, do not wait, but operate at once, and evacuate the pus through the perinæum. If you leave it to Nature, all may go well, but on the other hand, you may not be so fortunate, and the abscess may burst into the rectum, giving rise to a bad form of rectal fistula.

In all inflammatory conditions about the neck of the bladder, there is nothing better than rest, hot baths, and plain diet. Leeches to the perineum may be ordered, but hot water enemata generally afford much relief. Suppositories in the "Enule" form of morphia and belladonna have proved most effectual in my hands.

With regard to tubercular deposit in the prostate, I am sorry to say it is not often limited to this gland, but commonly affects one or both epididymes, also possibly the vesicles, urinary bladder, and kidneys. Can anything be done for this? When the deposit forms a small abscess in the prostate, massage might be tried; but I hardly think it worth while to advocate any extensive operations on the prostate for the removal of tuberculous deposit, when other organs are involved, such as the vesiculæ seminales, testicles, and bladder. A very large operation would be required for such a condition, and I hardly think the patient would consider "the game worth the candle."

Gouty deposits are not unknown in the prostate. Do not forget this when, in making a rectal examination, you come across a nodular prostate.

We now come, perhaps, to the more important part of our subject, namely, affections of the prostate in the aged. Amongst these the well-known hypertrophy of the prostate stands out pre-eminent. Then follow tumours, calculi, and malignant disease of the prostate. In hypertrophy of the prostate one must include adenomatous forms, because many cases of prostatic hypertrophy are purely outgrowths of the glandular elements, forming true and simple adenomata. Symptoms of enlarged prostate may come on at a much earlier age than is usually considered to be the case, for one has met with a prostate sufficiently large to give rise to symptoms before the age of fifty. It used to be said that you get no hypertrophy of the prostate before sixty. I have seen several cases myself as young as forty-five. It is said that enlargement of the prostate has some connection with excessive marital relations. We know that the prostate is connected with the sexual system more than with the urinary, and so it is quite possible that this may be the case. Enlargement of the prostate comes on very insidiously. The patient may notice first that he has to pass water a little more frequently than usual, that he cannot hold his water at night so

long as he used to; then he will notice that the stream is not projected with the same force; and in addition to that he may perceive a slight sense of discomfort, and later there is a sense of weight in the perineum. In more advanced cases there is a real difficulty in the outflow of urine, until at last it becomes a mere dribble. This is the stage before the final, which is absolute retention. An old man who is the subject of enlarged prostate may be quite ignorant of the fact until some day or other he commits an indiscretion; he may eat too heavily, or drink too much, or catch a cold, or all three combined, and the result is he gets a certain amount of 'congestion of his enlarged prostate, which will often give rise to complete retention of urine, for which he calls in the aid of a surgeon. Sir Henry Thomson has pointed out that the various elements of the prostate may separately be the chief seat of the trouble. For instance, there is the glandular tissue of the prostate, which may be hypertrophied, or there may be a hyperplasia of the fibrous or muscular element of this gland. All these elements may be increased possibly in certain cases in the same ratio; in others one or two may be increased at the expense of the third; and, according to the kind of prostate you are dealing with, so must you plan your operation, if an operation is called for. An enlarged prostate may grow in two different ways; it may be very discernible to the finger in the rectum; but, on the other hand, it may not be so evident there, and yet the patient may suffer more from urinary obstruction than does one whose prostate is markedly enlarged per rectum. As you know, the intra-vesicular portion of the prostate is the one which chiefly gives rise to troubles of micturition, whether it is the lateral lobes, a projecting third lobe, or a general collar-like enlargement which is chiefly at fault.

There is a condition called the bar at the neck of the bladder, which merely means a raising up of the fold of the mucous membrane with a little submucous tissue in it between the enlarged lateral lobes, and occupying the site of the so called third lobe.

The treatment of the enlarged prostate may be considered from a medical, as well as from a surgical, point of view.

It is some time since I was recommended to try a prescription for a suppository, which

emanated from some hospital surgeon fifty years ago, and which seems to have been forgotten. I have tried it with very good results. It consists of iodide of potassium combined with conium and either hyoscyamus or belladonna. The chief ingredient in it is the iodide of potash. To start with, one gives iodide of potassium 1 gr., extract conium 5 gr., extract hyoscyami 5 gr. That, given in the form of a suppository night and morning, is an excellent thing for reducing the size of a large prostate. If the patient—*i.e.*, his rectum—can stand this one grain of iodide of potassium, you may increase the dose, giving him up to five grains of iodide of potassium in a suppository, though so large a dose of the iodide is likely to cause irritability and a constant desire to defæcate. Combined with this I like a little nux vomica by the mouth, for where you have obstruction due to an enlarged prostate a certain amount of atony of the bladder ensues, for which nux vomica or strychnine are good. The use of these suppositories may often postpone operation for some length of time. I am not sure that one is doing much real good by postponing operation, because I think that where operation is called for in prostatic cases the sooner it is done the better. But the suppositories certainly enable "the evil day," as the patient calls it, to be postponed a little.

We must now consider catheterism. The doctor is called in to relieve retention due to an enlarged prostate, and, of course, he passes a catheter, and he may enjoin the passage of it daily, or even more than once a day, according to the urgency of the symptoms. If he has experienced much difficulty in getting the catheter in he may consider it better to leave it in for a time. What are the best forms of catheter to use in cases of enlarged prostate? On the whole, I think the best with which to commence is the coudée or bi-coudée catheter. Next comes the old gum-elastic catheter with a stilette. This is about the only thing that particular make is good for; it has become an antiquated weapon altogether, but it is good in some of these difficult prostatic cases. You should select a good-sized one—No. 12 English. In prostatic cases it is a mistake to use too small an instrument; the larger the instrument you use the better, and the easier it goes in. Should you meet with an obstruction at the neck

of the bladder it is owing to the enlarged prostate, it is not due to the size of the instrument. Why do I recommend the somewhat antiquated gum-elastic English catheter? It is because by partially withdrawing the stilette you tilt up the end, thus enabling the point of your catheter to ride over the obstruction. I have often seen this succeed when other means have failed. There is another manœuvre which may aid one in passing a catheter in cases of enlarged prostate. I refer to the aid which a finger in the rectum may afford if pressure is made upwards towards the bladder as the point of the instrument passes through the prostatic urethra.

The next most simple method of treatment which we come to is dilatation by metal bougies, not gum elastic. Here are some metal bougies which were made for that purpose. You will notice they are very much bellied at the elbow. I have no doubt that these, passed daily, would wear a sort of tunnel along the floor of the prostate. They are said to have done a certain amount of good, but I cannot say that I recommend them very strongly. The larger-sized metal bougie you use the better, and usually the patient experiences considerable benefit for a time. You will notice that we are gradually coming to operative interference. Where there is great prostatic irritation and discomfort, the mere passage of an instrument being annoying to the patient and attended with difficulty, and possibly with elevation of temperature, I think electrolysis, such as is advocated for stricture of the urethra, not at all a bad thing to try. Here I show you the electrodes which I have used in the treatment of stricture of the urethra. They consist of a metal tip with insulated shaft, and the negative pole of the battery is attached to this metal bougie, the positive pole being placed somewhere in contact with the patient's body—preferably as a pad under the sacrum. Patients can stand about five to ten milliampères without undue discomfort, and this pole is moved up and down the prostatic urethra. It allays spasm and irritability, and at the same time may, to some extent, act as a dilator.

Now we pass on to the operative treatment of the enlarged prostate. I will first mention the operations on the adnexa. White, of Philadelphia, drew the attention of the profession to the fact that castration was often followed by prostatic atrophy.

Other workers in the same field have followed, and now it is an established fact that not only amongst animals, but also in men, the removal of the testicles is often followed by atrophy of the prostate. If only one testicle is removed, atrophy takes place on the corresponding side. Castration not being an agreeable fate to contemplate, vasectomy or division of the vas deferens has been practised as a substitute. If the vas deferens is divided on one or both sides, you are supposed to get the same effect without some of the risks and without that sentimental objection which attaches to the other. Not only is there a sentimental objection attaching to castration, but there is a real danger of insanity following. It is also said that a loss of mental equilibrium may follow vasectomy, but I am happy to say I have not seen a case of that kind, though I have seen many cases of vasectomy. It is a very simple operation. One can readily feel the vas deferens through the scrotum, and it can be so fixed between the finger and thumb that no assistance is required. There are several ways of obliterating the vas, but the one I prefer is by crushing it by means of a modified Pollock's hæmorrhoidal clamp. By this method you avoid the use of ligatures. It is always well to avoid the use of buried ligatures in the scrotum, for they may give rise to after-trouble. I have known both catgut and silk ligatures applied when tying a varicocele set up suppuration in the bottom of the wound, which wound has only healed on the removal or casting off of the offending ligature. Always avoid the use of buried sutures or ligatures where possible in the scrotum.

What cases are best suited for vasectomy? You cannot expect to get much benefit in the large hard fibrous prostate. The prostate may be as large as you like, but in order to get the maximum amount of benefit it should be soft. A soft vascular prostate is the one best suited for treatment by vasectomy. I had a very good example of the benefit to be derived from this operation not very long ago. I was asked by Dr. Dobson to see an old gentleman æt. 65, who had been the subject of catheter-life for two years. On and off he had been bleeding in considerable quantities. I found the prostate to be enlarged and softish. I tried to see the condition of the intra-vesicular portion by the aid of the cystoscope, but there was too much

bleeding. As he was under an anæsthetic for that purpose, I took the opportunity of doing a double vasectomy; that is to say, I divided the vas on both sides at the same sitting. A catheter was tied in for a time, as there was so much bleeding, and I advised Dr. Dobson to wash out the bladder on account of hæmorrhage and the liability to the formation of blood-clots. This he did. It appears that the hæmorrhage ceased soon afterwards, that is to say, forty-eight hours after the operation, and now, a fortnight after the operation, he uses a catheter only twice in the twenty-four hours. The report says "he can pass a wineglassful of urine without aid. Yesterday in all he passed half a pint." His doctor says, "The great point in the case is that the patient has lost the urgent calls to micturate which rendered life miserable to him. He can now hold his water with comfort for six to eight hours."

The prostate has been a very troublesome gland to deal with, and all sorts of operations have been devised for its partial or complete removal. Bottini, of Italy, invented this instrument which I show you, for using with the galvano-cautery. It has a cutting blade, and is shaped like a lithotrite. The instrument is passed into the bladder closed, it is then reversed, and the blades so opened that the female blade hugs the back of the prostate; the small or male blade is then pushed forward and through the prostatic fissure, cauterising as it goes, and this process is repeated once or twice. There is a double channel for the circulation of water to keep the parts cool. This instrument is spoken of very well in Italy, and in certain other parts also. We tried it in London but got very little substantial benefit from it, and its use is not without risk. I prefer an instrument such as the one I now show you, which is the outcome of Mercier's inciseur, or a knife by which he divides the bar at the neck of the bladder. Gouley's prostatome punches out a piece the size of a pea. No doubt it would be a more efficient instrument if it removed a larger portion of prostatic tissue. I have treated several cases with this, and they have not given me anxiety; they were all partially successful for a time, but they rapidly relapsed. The instrument does not seem to do enough, and on repeating its use I have not derived as much good from it as on the first sitting. Having tried it in about half a dozen

cases I have now practically discarded it. The instrument is an interesting one. It is passed closed as if it were a lithotrite, and then inverted. The beak is passed behind the enlarged prostate, and then the male blade is withdrawn. A stilette or pin is now projected, which transfixes the gland and holds it in position. Then down comes this male blade, and when it has firmly grasped the obstructing lobe the instrument is locked, the screw power put on, and the piece is crushed off, coming away in the instrument.

Let us now consider supra-pubic prostatectomy, or McGill's operation. This operation is just now attracting a good deal of attention, and I must say the results I have seen seem to warrant it. McGill spoke highly of it some years ago, and he showed several successful cases at a meeting of the British Medical Association. A colleague of mine told me he saw these cases sitting in a row, each man with a plate on his knees on which was placed his prostate! After this the operation fell into disuse for a few years. I think surgeons were afraid of the amount of bleeding that might ensue, nor had they at that time the appliances we now have for bringing into view the bladder base. In some cases it is easy to shell out an enlarged prostate through a supra-pubic wound in the bladder; but in other cases it is quite a different matter. It depends so much upon the pathological condition present.

I will read to you the notes of two cases on whom I have recently operated. Here is a specimen which has been in spirit for two or three months, and therefore it is not as large now as it was when removed. It is from an old man of sixty, who was admitted to St. Peter's Hospital suffering with difficulty in micturition which at times amounted to retention. Even when the power of micturition was not in abeyance, it was found that he was unable to empty his bladder, ten ounces of urine remaining. On a cystoscopic examination under ether, the prostate was seen to be much increased in size, projecting into the bladder in two well-marked lobes which were separated by a deep sulcus. As it seemed to be a favourable case for removal of the prostate by the supra-pubic route, the following operation was performed: The urine having been drawn off and the bladder well washed out, the organ was distended with a solution of boracic acid. The

patient was not placed in the Trendelenburg position, nor was a Petersen's rectal bag employed. The bladder being opened in the usual way through a supra-pubic incision, a finger was inserted and the diagnosis confirmed, for it was found that the lateral lobes were much hypertrophied and projected into the bladder. A caisson tube was now passed in, and under the guidance of electric light an incision was made into one of the lobes, dividing the prostatic capsule. The caisson being withdrawn, a periosteal elevator, guided by the finger, was passed through the incision into the prostate, and by its aid one half of the gland was shelled out. The same manoeuvre was then repeated on the opposite side with a like result. The glandular tissue thus enucleated equalled in size a small Tangerine orange. It was more adherent than in some cases, and required a good deal of force for its separation. There was considerable bleeding, so that it was thought advisable to tampon the bladder with strips of gauze which, together with drains, were left in. I used an ordinary periosteal elevator, but found it too short for the work. So I have had some prostatic enucleators made by Mr. Montague, which answer the purpose well. This patient was operated upon on March 15th this year. In May he came up to the out-patient department, passing perfectly clear urine without difficulty or pain, and I found he completely emptied his bladder.

Here is another case very much on the same lines as the other. It refers to an enlarged middle lobe. This, again, was well seen by cystoscopic examination. The patient was admitted to the hospital with the usual signs of difficulty in micturition and dribbling, and now and again he had attacks amounting almost to complete retention. It was found that he had about ten ounces of residual urine. An enlarged third lobe was diagnosed by means of the cystoscope. On January 4th, I did a supra-pubic prostatectomy, and the finger passed into the bladder felt a rounded sessile third lobe. A caisson tube was introduced, and I made an incision into the prostatic capsule under the guidance of the eye with an electric lamp on the forehead. I then endeavoured to shell it out by means of a periosteal elevator, but unsuccessfully. I therefore grasped it with this pair of forceps and twisted it off. I did not get

it all away at the first attempt, but was successful at the third essay. Of course it spoilt the specimen, but on looking afterwards with the electric light I found I had completely removed it. With a little perchloride of iron on some lint I swabbed the prostatic wound, and completed the operation by inserting a large drainage-tube. I always use a tube in these cases, and usually employ two red rubber catheters sewn back to back and passed into the bladder, and in addition a large red rubber drainage-tube, so that there may be sufficiently free drainage to carry off any blood clots. He has now only one ounce of residual urine. Enucleation was futile in this case as the tumour was more fibrous than glandular; it was so tough that I had a good deal of difficulty in getting it away. As to the drainage-tubes I generally remove the first one at the end of twenty-four hours, and the double catheter, unless I want a permanent supra-pubic drain, at the end of forty-eight hours. The bladder then usually closes within a fortnight. This is an operation which I think will come more into prominence in the immediate future.

A colleague of mine, a week ago, shelled out a larger prostate than the one I show you quite easily; being an adenoma of the prostate it lent itself to this mode of treatment. Where you have pure adenomata of the prostate to deal with, the operation is surprisingly easy and the cases do very well. By the old perineal operation, portions of the prostate have come away during the performance of ordinary lateral lithotomy. Surgeons therefore thought they might tackle these growths of the prostate by means of a perinæal operation. But as a matter of fact it is a very difficult operation, and one which I should not recommend. It is often, indeed, difficult to get the tip of your finger into the bladder through a perinæal incision when there is an enlarged prostate, owing to the resistance of the tissues and the length of the urethra. You know in cases of prostatic hypertrophy, the prostatic urethra is considerably elongated, and may measure $3\frac{1}{2}$ inches in length. It has been pointed out that perhaps the two operations, viz., that of supra-pubic and perinæal prostatectomy might be combined, and it is not at all a bad suggestion, where the prostate is enlarged rather towards the rectum than towards the bladder. A transverse or curved incision, with the convexity

forwards, is made across the perinæum, a metal bougie or sound being held in the urethra. Through this incision, the prostate can be well exposed, more especially if the bladder is now opened supra-pubically, and pressure downwards is made by an assistant's finger placed on the vesical aspect of the prostate, which brings it down well into the perinæal wound and much facilitates perinæal prostatectomy. I have not done it, but I have heard it described, and I can quite understand its value in cases such as I have indicated.

Treatment of Abscess of the Lung.—Bell describes in the *Montreal Medical Journal* for May, 1901, some points of interest in three cases of abscess of the lung.

The abscess in each case followed a pneumonia, and judging from the symptoms probably began as a local gangrene of lung tissue. The abscess communicated with the bronchi, with the result that evacuations of horribly smelling pus, at first more or less intermittent, occurred, and continued until the cavity was drained. In each case there was considerable difficulty about the anæsthetic, and in one case general anæsthesia was impossible on account of the filling of the bronchi with pus, which interfered with the breathing, and produced violent spasms of coughing. In this case the attempt to administer ether, which was only maintained for a few minutes, kept the patient in a condition of semi-asphyxia and violent coughing for most of the afternoon. In each case the lung was firmly adherent to the chest wall, making the operation almost as simple as that for empyema, and finally the results of draining the cavities were most satisfactory. Localisation of the abscesses alone was difficult, the aspirating needle failing to give evidence of pus even when it had undoubtedly entered the cavity. This is easily understood when it is considered that such a cavity is necessarily at times more or less completely emptied by a fit of coughing, and that its contents are moreover thick and viscid, containing generally much mucus and some shreddy material, especially if the case is a comparatively recent one.—*The Therapeutic Gazette*, November 15.

MYASTHENIA GASTRICA: ITS DIAGNOSIS AND TREATMENT.

By GEORGE HERSCHELL, M.D.Lond.

(Continued from page 144.)

TREATMENT OF MYASTHENIA WITHOUT RETENTION.—That is, of the first or second degree.

It is needless to point out that if we can find any obvious cause for the myasthenia, such as habitual overloading of the stomach, chronic gastritis, neurasthenia, or an affection producing portal congestion, we must first attack these *secundum artem* before we can reasonably hope to benefit the local condition. It would, however, be out of place in this article to recapitulate the well-known methods of treatment in such cases. Consequently only those special methods of treatment will be discussed which have for their object the restoration of tone to the myasthenic stomach.

Diet.—The first essential is the regulation of the food which is consumed by the patient. The main points are: 1. All food which does not undergo digestion in the stomach must be prepared in such a manner that the weakened organ can with facility expel it through the pylorus. It is not, therefore, so much the kind of food which is of importance, as the condition in which it is presented to the stomach. In the first degrees of myasthenia, therefore, where there is no absolute retention of food residues, and consequently no fermentation to speak of, we need not exclude articles of diet such as sugar, and we may give eggs, roast or grilled meat *ad libitum*, and trust to mastication and digestion in the stomach to reduce it to such a condition that it will easily pass through the pylorus. With other food substances it is far different. Bread should be cut into thin slices and well toasted, in order that the envelopes of the starch granules may be ruptured and its further digestion facilitated. An additional advantage will be that the lactic acid germs which always exist in every part of a loaf of bread will be destroyed. Any one may demonstrate for himself the presence of lactic acid germs in ordinary bread by soaking a little of the crumb in warm water and keeping it for a few hours in a thermostat. The presence of lactic acid can then be readily demonstrated by the usual tests. All green vegetables and potatoes should be in the form of a purée, that is to say,

boiled and then passed through a sieve. Such vegetables may be made much more palatable by stewing them in some good veal broth with a tiny piece of bacon or ham to give a flavour. Endive and lettuce, vegetables rarely seen cooked in this country, are exceptionally delicate and palatable when treated in this manner and made into a purée.

2. The other main point in the diet is the management of the liquid taken by the patient. Since the chief characteristic of the myasthenic stomach is its inability to easily empty itself of fluids, we should study to lighten its work in this direction as much as possible. To this end we absolutely prohibit soup, limit the amount of fluid taken at any one meal to four or five ounces, and direct that the remaining liquid necessary for the carrying on the functions of the body should be taken when the stomach is presumably empty, about an hour before meals. Mathieu* suggests that an effectual way of limiting the quantity of liquid consumed by the patient is to order it to be taken as hot as possible at the close of the meal. The patient has no temptation to take it to excess, and he believes that it exercises a contracting effect upon the stomach. We cannot do better than quote his own words: "On donnera du thé léger, du grog très léger fait avec du cognac, ou du rhum, en nature, ou préférablement brûlé. Certains malades préfèrent la camomille, la décoction de fleurs d'oranger, de fleurs de tilleul, ou d'autres décoctions plus ou moins agréables au goût. Qu'importe? Le but est en somme de faire de l'eau chaude et de la rendre acceptable. . . . Pour notre part, nous laissons les malades soumis aux boissons chaudes boire à discrétion. Ils n'ont dans ces conditions aucune tendance à boire avec excès, et les faire boire chaud équivalent généralement à les faire boire moins."

This limitation of fluid will usually suffice in the milder degrees of myasthenia; but, as we shall presently see, when there is permanent retention of food residues, a considerable proportion of the fluid necessary for the maintenance of the body must be given by the rectum.

TREATMENT APPLIED LOCALLY TO THE STOMACH.—At the same time that we are directing our therapeutic efforts to the removal of the cause of the myasthenic condition, if discoverable, we

* A. Mathieu, 'Thérapeutique des Maladies de l'Estomac,' 3me édit., Paris, Doin, 1898, p. 168.

should proceed *pari passu* with direct local treatment of the stomach itself. Thus, whilst we are treating a failing and dilated heart with digitalis, carbonated Nauheim baths, or resisted movements, or curing a general neurasthenia with static electricity, we should systematically attack the stomach locally with the weapons which modern science has placed at our disposal. We have two chief methods of acting directly upon the stomach, viz. massage and electricity. We will discuss these *seriatim*.

Massage: manual and mechanical.—Of ordinary manual massage very little need be said. Theoretically it should be of the greatest use, practically it usually fails to achieve any useful purpose in the treatment of gastric atony. The reason is not far to seek. In the hands of Zabładowski ('Berlin. klin. Wochenschr.,' 1886, No. 26) and other physicians skilled in the special technique of emptying the stomach by manipulation, it has produced brilliant results. These, however, are not reproduced by the half-educated and imperfectly trained women into whose hands the practice of massage in this country has, unfortunately, been allowed to drift. Nor could we reasonably expect anything else seeing that destitute of any practical acquaintance with anatomy, their slender professional knowledge is, in most cases, limited to some purely routine methods acquired by a few weeks' attendance at a school of massage.

A vastly superior agent for general use is to be found in the mechanical massage produced by rapid vibration. The use of mechanical vibration in the treatment of disease is not by any means of recent introduction, dating back as it probably does to 1724, when l'Abbé Saint Pierre invented his "tremoussoir;" Ling in 1813, Zander in 1864, further improved the methods. The first really important work was, however, that done by Vigouroux in 1878. After him, Boudet and Mortimer-Granville devoted considerable attention to the subject, and were followed by Braun and Trieste in 1890, and Charcot in 1892. Since then Jayle and de Lacroix have used this method extensively, and it is to the last physician that we owe its modern term, "sismotherapy" (from the Greek words *σεισμός*, a shaking, and *θεραπεία*, therapy).

A considerable experience leads me to the conviction that with the exception of electricity, this method of treatment is the most valuable means which we have at our disposal for restoring tone to the gastro-intestinal tract. It has, moreover, the great advantage over ordinary massage that the practitioner can, and indeed should, apply it personally, and thus keep the treatment of the case in his own hands and not depute it to others.

Until recently the best apparatus to be obtained was the Swedish vibrator of Liedbeck, actuated by means of a small electro-motor. It has now, however, been practically superseded by Dean's sismogen,* an instrument vastly superior, not only to this, but in my opinion, to all the other apparatus for producing vibration at present procurable. The sismogen, as the word indicates, is a generator of vibration, and differs from all other vibrators in design and capabilities. The constructor of this machine has incorporated in it the best engineering methods, and has made an entirely new departure in these machines. The Dean sismogen produces both centrifugal and centripetal vibrations without addition to its parts, but by a simple change of position of the rod carrying the applicator. This is well shown in the accompanying illustration (Fig. 1). In the older instruments, such as the Liedbeck vibrator, the oscillation is produced by the usual concentric mechanism, but the sismogen depends for its powers of vibration on a mass of mercury revolving eccentrically within a sealed chamber attached to the handle. The handle is connected to a revolving flexible shaft encased in a metal sheath or armouring. This flexible shaft is attached at its other end to an electro-motor of suitable power and under the usual control. The sismogen in operation produces centrifugal vibration when held in the hand with the applicator in concentric position, and imitates in a truthful manner the massage movement known as kneading. The depth or degree of the vibratory movement is produced by the pressure applied by the operator, and may be of a light or skimming order or may be a profound impression reaching the whole organism. When the applicator is turned at right angles with the handle, the movement imparted is

* De Lacroix, 'La Sismotherapie,' Paris, 1899, p. 11.

* A. Dean, 73, Hatton Garden, E.C.

that of knocking or percussion, but of a slightly circular order. The machine possesses a further advantage in its mechanical arrangement, which consists of a universal joint which allows the flexible shaft to hang down without danger of breaking off the stranded cords. In all other machines this point is neglected, and the owner often has to renew his flexible shaft.

should be first applied to the cardiac region of the stomach and moved towards the pylorus. When this is reached the applicator is removed and replaced upon the cardiac end of the stomach. Light vibration over the solar plexus for a few minutes may conclude the séance. The treatment at first should not be longer than five minutes, but the duration can be gradually increased until

FIG. 1.



The best time for applying vibration to the myasthenic stomach is about an hour before meals. It will thus assist the expulsion of the last digested meal into the duodenum, and prepare the stomach to receive the approaching one. With this end the applicator of the sismogen

it lasts from fifteen to twenty minutes. I usually apply vibration and electricity upon alternate days.

Electricity.—We have two currents with which we may restore tone to a myasthenic stomach—that from a high-tension induction coil apparatus, and the sinusoidal current.

I may premise that by saying that the small faradic apparatus commonly met with in the hands of nurses and masseuses is absolutely useless in the treatment of diseases of the stomach. Consisting as it does of a very few yards of wire in the secondary coil without means of regulating the electromotive force of the primary, and without rheostats in the secondary, circuit, with an interrupter giving a harsh and uneven vibrator action, it cannot be considered a therapeutic instrument, and must be absolutely useless for any scientific form of treatment.

In the hands of experts these have been entirely

fluous. We have here everything which the expert may require, but nothing which could be safely omitted. The apparatus consists of a box containing the coil and six dry cells to work them. In front it forms an upright switch-board. The coil is a compound one containing 1500 yards of No. 88 wire, 800 yards of No. 32 wire, and 238 yards of No. 21 wire in the secondary coils. In all, 8000 feet of wire in the primary and secondary coils in 15,000 windings. At the top of the switch-board (see Fig. 2) is a compound switch by means of which different lengths of wire may be selected for use and switched into use circuit. On the right

FIG. 2.



superseded by what are known as high-tension apparatus. By high tension we understand a current with a relatively high voltage and small amperage, and which requires for its production a coil of very long thin wire. One of the best and most perfect pieces of apparatus of this kind is that manufactured by the Jerome Kidder Company of New York from the design of Professor Monell, and as this may be taken as the type of what such an instrument should be, a description of it will best show my readers what are the essential features which a practical therapeutic coil should possess. And I may say that nothing is super-

hand side a rheostat in the primary circuit enables the electromotive force from the six cells to be graduated. On the left are a couple of liquid rheostats containing an unalterable fluid of known resistance, which enable a resistance of 1,000,000 ohms in the first tube and 55,000 in the second to be introduced into the secondary circuit and gradually withdrawn. We have thus the power of absolutely regulating the current which reaches the patient without affecting the rapidity of action of the interrupter. Rapid and slow interrupters, a pole reverser, and a moveable magnetic field complete this magnificent piece of apparatus. In this

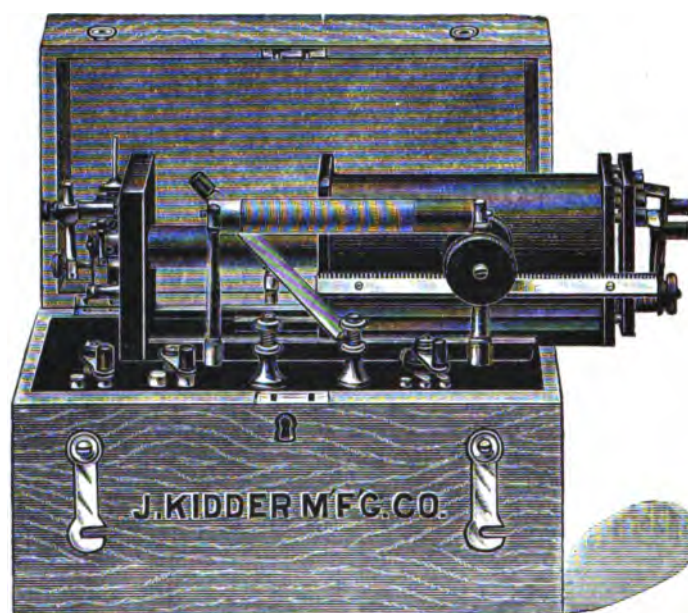
machine the secondary coil is fixed to entirely cover the primary, since, unless this is the case, the full benefit of the great length of wire is not obtained. In another and more portable pattern • (Fig. 3), the secondary coil is mounted on a sledge worked by a rack and pinion, and the regulation of the current strength is obtained in this manner. In this pattern the rheostats in the secondary circuit have been sacrificed to portability, but in other respects the apparatus is identical.

The current from the induction coil apparatus may be applied percutaneously or internally.

By external electrodes.—One large electrode is

the epigastric region. The current strength to be used is such that good contractions of the abdominal muscles are produced at each interruption. For treating gastric myasthenia the slow interruptions should be used. Rapid interruptions would produce a sedative action, and, whilst appropriate for the treatment of hyperchlorhydria or gastralgia, would fail to benefit muscular weakness of the stomach walls. The best electrodes to use for application to the abdominal wall consist of a square or circular piece of metal covered with a piece of felt or sponge and backed with a piece of sheet indiarubber projecting an inch all round.

FIG. 3.



placed over the stomach and the other upon some indifferent part, for preference the gluteal region. Eight hundred yards of No. 32 wire are switched into circuit, and two or three cells brought into action. The rheostat in the primary circuit is now adjusted in correlation with the vibrator screw and the magnetic field to give with the slow interrupter about 200 interruptions a minute. The electrodes are to remain stable until the end of the séance.

By one internal and one external electrode.—

Boardman Reed's gastric electrode is introduced into the stomach, the patient having previously swallowed a tumbler of water. The large pad connected with the other pole is placed as before upon

The other current which is useful in the treatment of myasthenia of the stomach, the so-called sinusoidal current, has the advantage that it is much less painful and disagreeable to the patient.

In the ordinary induction coil apparatus the current which is applied to the patient is that which is induced in the secondary coil at each make and break of the interrupter. And as this make and break takes place quite suddenly, it follows that the induced current will reach its maximum instantaneously, and with equal rapidity will fall to zero. If you pass a coil of wire through the magnetic field about one pole of a magnet, a current will be induced in it, but will gradually

reach its maximum and as gradually return to zero. If such a coil in the form of a semicircle revolves around a centre placed between the poles of a magnet the induced current will be alternately + and —, and such a current is termed a sinusoidal one, as the phases are equal, and the comfort of its application follows from the fact that the alterations of potential are gradual and not sudden as that from the induction coil apparatus. The sinusoidal generator for sinusoidal voltaisation may be of two or more different forms. In some cases it may be convenient to employ the alternating current supply mains and with certain electrical arrangements to transform down; this may be done with perfect safety, but these currents are rarely of a true sinusoidal nature, owing to the commercial machines

induction coil. In fact, the patients chiefly feel the pull of the contracting muscle. The muscular contractions produced by it are very deep and spreading.

In treating myasthenia of the stomach we apply the current with two objects and in two manners. We may attempt to influence the metabolism of the patient and the innervation of the stomach through the sympathetic ganglia, or we may act directly upon the nutrition of the gastric muscularis.

With the former object we apply one electrode to the epigastric region. This may be attached to either pole. The other pole is connected by a bifurcated cord to two long narrow electrodes, one of which is placed along each side of the dorsal vertebræ.

FIG. 4.



being deficient upon this point. The most suitable and efficient plan is to use a direct current generator or motor having its armature tapped at equal distances and connected by wires to slip rings upon the axis. From the brushes which are in eccentric connection with these rings a current is taken off of a true sinusoidal nature.

The apparatus which I have used for some time is of the form provided with permanent magnets, and is actuated by a small continuous current motor on the common axis. This is wound for 110 volts, and is run from the house main. The makers are the McIntosh Battery and Optical Company, of Chicago. It is shown in Fig. 4. It produces a remarkably smooth sinusoidal current which is much less painful than that from an

It is important to bear in mind that the combined areas of the two dorsal electrodes must equal that of the anterior one. We can manage this by having the former 4 inches by 2 inches in size and the latter 4 inches square. This will give us an area of 16 square inches in each case. In choosing the position for the posterior electrodes I follow the practice of Dr. Henri Guimbail, who pointed out that at this spot the sympathetic ganglia are particularly accessible to the current.

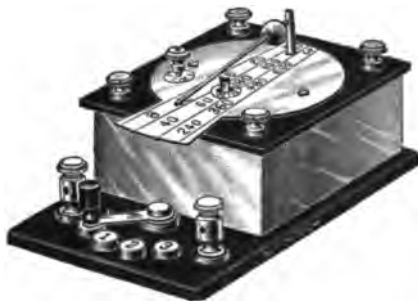
"Je fais cette application au niveau de la région dorsale parce que c'est particulièrement en ce point que la chaîne ganglionnaire est accessible au courant. Aux régions cervicale, lombaire, et sacrée, le sympathique est situé au-devant de la colonne vertébrale; la difficulté de l'atteindre est

considérable, tandis qu'à la région dorsale il est appliqué en arrière de la plèvre, au-devant des articulations costo-vertébrales. Le plus souvent les ganglions se trouvent en rapport avec les trous de conjugaison, ce qui les rend encore plus directement accessible." *

For this application the current should be very feeble. In fact, of such a strength that it is only just comfortably felt by the patient.

To act directly upon the muscular substance of the stomach we require to give the current strong enough to produce efficient muscular contractions. But it is important to remember that the mere fact of causing a muscle to contract by means of an electric current will not of necessity improve its nutrition. If we apply the current from an induction coil apparatus or from a sinusoidal machine to a muscle, this will be thrown into tetanic contraction which will continue until the

FIG. 5.



electrode is removed. or until the excitability of the muscle is exhausted. And it must be obvious that we cannot restore tone to a muscle by thus exhausting it. Unfortunately, this is what is usually done by nurses and other untrained persons who attempt to apply electricity. They simply apply the current to a muscle and keep it in a state of contraction for an indefinite time, until the spirit moves them to shift the electrode on to another one. They thus aggravate the condition of weakness which they are attempting to cure. As a matter of fact, the proper technique for strengthening a muscle is to produce short contractions, followed by periods of rest. We thus cause the muscle to perform work which is well within its capacity, and allow it time to recuperate before the next effort. With a modern high-class induction coil apparatus this can be

done by means of the slow interrupter, with which these batteries are provided. With the sinusoidal apparatus we must make use of an accessory piece of apparatus known as a rheotome, which is constructed on the principle of a metronome, and breaks the current a given number of times a minute. One of the best is shown in Fig. 5, and is actuated by clockwork. The number of interruptions a minute can only be learnt by experience, and will depend upon several factors.

The duration of the treatments with the sinusoidal current should at first be very short, the length of the séance being gradually increased as the tolerance of the patient is ascertained.

The possession of the McIntosh apparatus is of extreme value to any physician who is often called upon to treat cases of neurasthenia, as by its means may be given the sinusoidal baths, which are so useful in this condition.

It is needless to say that the application of the sinusoidal current cannot be deputed to a nurse or to the patient himself, as its successful use demands not only considerable experience in the medical application of electric currents, but also a practical acquaintance with the handling of dynamos and electro-motors.

Accessory measures.—There are certain other procedures which, although of minor importance, must not be neglected in the management of gastric myasthenia.

In many cases the stomach may be assisted to empty itself by placing it under better mechanical conditions. It can be readily understood that a stomach which will give way, and stretch under the weight of the contained food, is working at a considerable disadvantage when it attempts to empty itself through the pylorus. The stomach will have to raise its contents through a greater space. And, as Sir William Broadbent pointed out some years ago, this difficulty will be accentuated by a kinking of the pyloric end of the stomach from the drag of the heavy and dilated stomach upon its ligamentous attachments. Such being the case, we can easily see how much help can be given to the labouring stomach by affording it some artificial support, and thus taking off some of the weight of its contents, and diminishing the mechanical work demanded from it. We may do this in several ways.

By posture.—We may cause the patient to assume the recumbent position for an hour or so

* 'La Thérapeutique par les Agents physiques,' Paris, 1900, p. 39.

during the height of the digestive period upon a couch, the foot of which has been raised upon blocks.

Abdominal supports.—These are of great value, and may be applied in the following ways :

(a) *Plaster strapping.*—This is especially useful in the case of hospital out-patients who can only attend once a week. The lower abdomen should be evenly strapped with inch-wide strips of india-rubber plaster. In order to obtain the best results the strapping should be applied whilst the patient lies upon his back upon a couch tilted in the same manner as for the treatment by posture. The viscera will thus tend to gravitate upwards towards the diaphragm, ptoses will be corrected, and the plaster applied so as to give real support. I have seen the dragging sickening pain of a myasthenic and ptosed stomach cease as if by magic after the application of such a binder.

(b) *Woven woollen bandages.*—In the case of private patients, who probably take a daily sponge bath, a bandage which can be removed is, for obvious reasons, to be preferred to the rubber strapping. To be a success the bandage must be specially woven in such a manner that it is elastic transversely but not longitudinally.* The bandage should be applied spirally round the abdomen, commencing well over the hips, and taking care that each fold overlaps the one below for at least half its width. Three safety pins on each side will fasten the folds together and effectually prevent the bandage from slipping upwards. The patient will readily learn to apply it himself in such a manner that it will keep in position all day, and this should be done every morning in the same recumbent posture as the rubber plaster. In cases of gastro-ptosis or floating kidney, a suitable pad may be applied under the bandage to give additional support.

(c) *Abdominal belts* for the purpose of giving support to the lower abdomen are to be procured at the different instrument makers, but I have yet to see one which even tolerably fulfilled its function.

(d) *Corsets.*—The very best support with which I am acquainted is the special corset of Madame Gaches Sarraute, M.D., 61, Rue de Rome, Paris. This lady has devoted her attention entirely to the subject of supporting the lower abdomen by means

of scientifically constructed corsets, and has written a very valuable monograph on the subject.*

It is impossible to lay too much stress upon the value of abdominal support as a routine treatment for the early stage of myasthenia gastrica. We may, in fact, by means of a properly applied bandage, do much to check the affection and prevent it from arriving at the final stage of retention myasthenia. One of the main factors in the production of this third stage is the kinking of the pyloric end of the stomach already alluded to. This is well put by Van Valsah in the following words: "Normally, the pyloric end of the stomach, as it becomes distended by the contents. . . . , forced into it by peristalsis, extends downwards and to the right, and is elevated by rotation forwards and upwards, thus relaxing the gastro-hepatic ligament, and making it easy for fluids to pass. In myasthenia the enlarged pyloric antrum simply descends in the abdomen, tightens the gastro-hepatic ligament, and produces an angular constriction which renders the evacuation of the stomach more difficult. If the patient be emaciated or the abdominal tension is low, the production of the duodeno-pyloric constriction is facilitated."†

Unfortunately in these cases several factors combine to reduce the tension in the abdomen—relaxation of the abdominal muscles, the absorption of abdominal fat when emaciation is present, and the general neurasthenic condition of the patient. We can therefore see how very important it must be to supply artificially that support which should normally be present to the stomach. It is for these reasons that systematic exercises for increasing the tone and strength of the abdominal muscles in the manner advocated by Sandow, Luis Phelan, and Schreiber are so very useful. The best exercise of all for increasing the strength of the abdominal muscles is to keep the abdomen drawn in by a conscious effort. After a time this will be done unconsciously and automatically, with a corresponding improvement to the intra-abdominal tension and nutrition of the muscles.

We now pass to the study of the third or final stage of myasthenia gastrica.

(To be concluded.)

* Such a bandage, under the name "lanoid," can be procured at Wallas, 9, Great Marylebone Street, W.

* 'Le Corset,' par M^{me}. Gaches Sarraute. Paris : Masson, Editeur, 120, Boulevard St. Germaine.

† Op. cit., p. 364.

**THE APPLICATION OF THE CURATIVE
EFFECT OF THE VIOLET RAYS OF THE
ELECTRIC SPARK (BY A NEW METHOD)
TO CERTAIN FORMS OF DISEASE,
WITH A SUGGESTION OF THEIR EMPLOY-
MENT FOR THE DISPERSION OF
CANCER AND OTHER FORMS
OF MALIGNANT GROWTH.**

By ERNEST KINGSCOTE, M.B., C.M.,
L.R.C.S. Edin.

THE growing interest in, and probability of, our scientists discovering in the not far distant future, some means, other than the scalpel, of controlling, and even dispersing, malignant growths, brought to my mind a note-book of 1895, wherein were recorded some very interesting phenomena and most encouraging results in the treatment of nævi, lupus, and papillomata.

The first case was one of conjoined capillary and cavernous nævus in an infant. The growth extended from the left inner canthus down to the ala nasi, across the cheek, and upwards in front of the pinna till it reached the level of the top of the lower lid, so that nearly the whole cheek was involved, and the left orbit was completely occluded by the nævoid condition of the lower lid.

The disfigurement was frightful, the growth, as is usual in these severe cases, becoming purple and black when the child cried. Ordinary galvanopuncture was tried, but failed.

A Schall's four-celled bichromate cautery battery giving a current of 20 ampères with less than 8 volts was employed. Two needles, varnished to within a quarter of an inch of their points, were then dipped in an antiseptic solution, and inserted at different parts of the growth, and their points approximated and rubbed together. After each contact of the needle with one another a spark was produced of intense brightness, sufficient to illuminate the tissues surrounding the needles with a bright roseate hue.

This recurred every time the needle points were brought into contact and separated. Although the needle points were fused no ill-effects whatever resulted to the patient.

After twenty sittings the growth entirely disappeared, gradually getting paler, and sinking down to the normal skin level.

Each sitting lasted twenty to thirty seconds, and was repeated bi-weekly. I have now before me a photograph of the patient, taken two years afterwards, in which it is impossible to make out any traces of the formerly existing nævus.

This method has since been tried in many cases of nævus, with uniformly good and even brilliant results.

It was then tried in intractable cases of lupus,

and was equally successful, and later in cases of papillomata, which it simply annihilated. When I gave up general practice, and came to London in 1896, I was anxious to place the matter in the hands of a reliable surgeon, who could carry out researches on a large scale, not only on lupus and nævus, but also on malignant disease. The late Mr. Cotterell (who was much interested in electricity) undertook to do this, but his regrettable and premature death put a stop to the many researches on which he was engaged.

Now why does this spark have such an extraordinary—almost magical—effect?

Finsen, of Copenhagen, has shown that lupus can be cured by the flooding of the diseased tissue with light.

He employs some 500 volts and 80 ampères, and thereby produces a great deal of heat, so much so that it necessitates the patient being removed some distance from the light, and the interposition of a water screen between the patient and the source of light.

The water-cooling arrangement, and the great distance between the patient and the source of light, must cause a great loss of light. Further, Finsen says that it is only the *violet rays* that are of service as germ-destroying agents, and that the red and yellow rays, which, with the violet, go to make up the constant stream of light, are inoperative.

The *violet rays* are fatal to many forms of animal organisms, and probably to low forms of cell life, such as go to make up embryonic tissue.

Now we come to our point. The light produced by the breaking spark consists mostly of *violet rays*, and by the above described method they are introduced into the very centre and seat of the disease.

These few lines are written in the hope that those willing and able to conduct a ruthless campaign against malignant disease may find a suggestion in the description of those methods which the writer has found so efficacious in the treatment of lupus, nævi, and papillomata.

As evidence of the cancer bodies of Russell observed by Plimmer, Metchnikoff, Sansfelice, Roncali, and others seems to be accumulating (these authorities only being at variance as to whether they be Protozoa or Blastomycetes), there would be the greater probability of the violet rays of the breaking spark affecting cancerous growths which may, possibly, depend for their existence on these germs.

As long as the needles touch a strong current passes through them, and a powerful spark appears when the contact is broken, but owing to the low voltage of the current employed, and the high resistance of the tissues, a very weak current only can reach the patient, and no shocks or pain are felt by him on breaking the current.

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A CLINICAL LECTURE

ON

FATTY TUMOURS AND GENERAL ADIPOSITY.

Delivered at the London Hospital,

By JONATHAN HUTCHINSON, LL.D.,
F.R.C.S., F.R.S.

GENTLEMEN,—We have changed our subject from the diseases of vegetables, about which we spoke last time, to the occurrence of increase of fat in the human subject. But I will give one little connecting link between the two which is of interest. I did not mention at the last lecture the occurrence of tumours, which are not very frequent, in some species of vegetables and trees, more especially in the beech tree, in which a little marble-like nodule develops under the bark, consisting of wood, and having no connection with the wood of the stem of the tree. Those who are familiar with the beech tree will have observed these tumours on the stem, and if he will cut one out, he will find it to consist of a marble-like piece of wood covered in with bark, that wood being in the bark and not in connection with the stem. What is the explanation of the development of this? I believe that in connection with the stem of a large tree there is developed a little bud, which would make a leaf bud, but which, owing to some deficiency in the supply of sap, and deficiency in connection with the tree, gets shut off, and covered in by the bark of the tree; its basis of attachment to the tree narrows, and in the end there remains only the bud. And here we have a proof of the vital tendency of tissues, in the fact that this piece of vitalised tissue, the bud, potentially continues, although it has no object to fulfil in connection with the tree, and it cannot develop into a branch, because it is no longer connected with the stem, but is buried in by the bark. But there it grows, it does not have leaves, but

forms a rounded mass covered by the bark. And this rounded mass will form a layer of wood. The very best developed wood could not be better than some of these detached nodules. I have a very beautiful specimen which I obtained a few weeks ago from a large fir tree: it did not need to be cut off, but was knocked off; there was no structural connection with the tree. The mass was as large as my two fists; and on making a section, one could see layer upon layer, just as you would in the stem of the tree. It is a curious instance of tissue which is endowed with vital tendencies; although it can no longer develop for the good of the individual as other buds do, producing leaves and organs in connection with the parent tree, still it cannot help going on growing, it is not absolutely dead.

My argument in reference to fatty tumours is, and as many of you know, it is an explanation which has been applied not only to fatty tumours but to other tumours, tumours of a malignant character, that a little bud of tissue becomes detached from the parent in which it is embedded; in this instance a little bit of fat gets disassociated from the general organism, and is no longer in structural continuity with the fatty tissue or cellular tissue in which it is embedded. This little bit of fat may be detached at a very early period of life, and may be encysted so that it no longer undergoes ordinary development into the tissues to which it belongs. It remains isolated, and at some period of the patient's life it may be destined to grow into cancer. You know that these partake more or less of the qualities of positive tissue. With regard to ordinary lipomata of fatty tissue, my suggestion is that in all probability, during the primary development of the infant, little bits of fat get detached and grow. They do not necessarily grow in childhood; fatty tumours rarely grow in childhood, they grow at some period long after childhood, and you may get them at any age. That is my suggestion as to the origin of fatty tumours, and it has a general application to other tumours as well. I think it is a plausible explanation. That leads me to this statement: that fatty tumours, ordinary lipomata, are isolated fatty tumours as a rule, and that they are detached from the adipose tissue in which they are developed. And that leads me to a very important rule in surgery as to how we are to deal with them. I

will not go into the general surgical relation of fatty tumours, and the diagnosis is perfectly easy to anyone who has devoted attention to the subject. You know that a fatty tumour has a *pseudo-fluctuation* because it is very soft, because the fat in the human body is semi-fluid. If you were to let the fat out whilst it was warm you would notice it was almost fluid. So you have to take care that you do not mistake a fatty tumour for one containing actual fluid. You press up the tumour so as to make its borders bulge, and you notice that at its borders a certain amount of lobulation occurs—that is the diagnostic point. The position is another help in the diagnosis. On the shoulders these tumours are very common. Almost on any part of the body fatty tumours are developed where there is fat. It is extremely rare on the scrotum on account of the non-occurrence of fat there; it is extremely rare in the orbit although the orbit is full of fat. That is curious. Although you have a pad of fat in the orbit there is no tendency to aberration of growth there. Perhaps that is because the fat in the orbit is serving a more definite physiological purpose than fat in other parts of the body; it serves the essential purpose of supporting the eyeball, and is less liable to vary in quantity than is the fat of other parts of the body. That is one reason why fatty tumours are so rare, and, indeed, are almost never found, in the orbital region. The assertion that fatty tumours are completely encapsuled, and have no structural continuity with the tissues in which they lie, has an important bearing in this way, that it is not then necessary to dissect them out; what you should do is to cut freely into the capsule in which they are embedded and in which they are perfectly loose, and then turn them out. You can turn them out easily if you open the capsule well. In enucleating your fatty tumour take your knife, squeeze the tumour up tightly, and then with your knife make one long sweep over the whole length of the tumour and cut it right out. If you attempt to cut round it you will have a long dissection. Its coat is quite fixed, but is itself perfectly loose; you have to cut freely and turn it out.

That is what I have to say respecting what are ordinarily called fatty tumours; but it is a very small part of the general subject, which I felt I could make some remarks upon—the conditions under which fat may vary in the animal organism.

We have the term lipoma for the isolated fatty tumour, and we have the term lipomatosis as designating the tendency to general increase of fat. We have several curious forms of this tendency to general increase of fat. I may take, first of all, a form which comes near to the isolated fatty tumour, but which just falls short of it. I have been in the museum but could not find a portrait of it, though I dare say all of you have seen examples in the hospital, namely, what is called diffuse lipoma. That is not diffuse lipomatosis, that is to say, it is not general lipomatosis. There is, in that condition, no tendency to general hypertrophy of the fat all over the body, but there is a tendency to hypertrophy of the fat in certain definite parts; at the back of the head is a common place; sometimes great cushions of fat form there. Great masses also form under the chin. They are the most common parts for these aggregations of fat. Next, they may form on the pubes, and on the scrotum. In this region the fat may be so collected into heaps that it may bury the penis. In lipomatosis the fat is not isolated as in the case of a tumour. It is an overgrowth or hypertrophy of the subcutaneous fatty tissues, and it is absolutely continuous with the fat of the surrounding parts. If you take one of these tumours away you will find it has not got a capsule. It is continuous with the subcutaneous fat, and you may dissect it away in a mass; the fat has no limiting membrane at all. Sometimes these masses are of enormous extent. Probably you have seen men in the streets; there is one man in the West End who is frequently having attention drawn to him. He has an enormous collection of fat, and everyone turns round to look at him. The neck is far more frequently affected with this condition than is the pubic region. I have only been consulted once in my life on account of the inconvenience of the fat in the pubic region. In that case there was a great mass which disfigured the lower part of the body, and the disfigurement was evident through the man's clothes. There was no tendency in the man to become corpulent, that is to say, he was not a very fat man.

These cases of local aggregation of fatty tissue occur, as far as I know, only in men; I have not seen a single case in a woman. And they occur definitely in connection with one cause, and that cause is associated with diet. Nearly always the

men who suffer from them are men who drink malt liquor very freely, and they have generally come to recognise the fact that they could restrain the growth of the fatty tumour and diminish its size very considerably by abstaining. But they are very often men who are not inclined to adopt that measure of restraint. I have had that testimony from several of them, that the tumour gets smaller if they leave off beer. From that we get this lesson, that the tendency to increase of fat is under the influence of diet, and therefore it is a general law that those people who have a general deposit of fat should abstain. You could scarcely have expected that diet could have had such a definite bearing upon these local growths, because I repeat that the growths are local, and they remain in the position in which they are discovered. They are not attended by any serious affections, but they are very disfiguring.

Ought we to operate in those cases? That will depend very much upon the patients. If the patient has tried abstinence, and has found that to fail, and if the disfigurement from the tumours is great, I think it is fair for the surgeon to undertake the operation. We have then to dissect a large mass of subcutaneous fat, and we have to cut into it. We shall not know where to end, because there is no limit to it; it merges gradually into the surrounding tissue. But you can cut away a great mass, and the unsightliness will probably be cured. The wound must be a very large one; perhaps it will be necessary that it should be one which will encircle the whole of the back of the man's neck. These large wounds of the skin are, I think, liable to a little increased risk as regards erysipelas compared with that attending a smaller skin wound. That is my experience, and I am a little cautious in urging upon a patient very extensive dissection of skin, because in spite of aseptic measures you are not always able to prevent erysipelas. A woman had it some time ago, and she did not recover from it. I have removed these tumours from the back of the neck, but not in one tithe of the cases I have seen. I have removed them in a few of the extreme cases, and the patients have been pleased with the result. In the case I have mentioned, in which the man had enormous masses growing from his pubes, covering up his penis altogether, and which were a great inconvenience to him, he

was admitted to London Hospital under my son's care, and he removed them. That man did have an attack of erysipelas afterwards. He has enormous masses on his neck still, with which we shall not meddle. He is engaged as auctioneer's assistant in a horse auction room, and these men, as a rule, take beer very freely. Finding that abstinence has a great influence in reducing the size, he has been drinking less.

I must just mention those who have chiefly written on this subject. I believe Sir Henry Brodie was the first, and Sir William MacCormac and I have recorded many cases.

We have now dealt with localised lipoma, which is encysted, and is due to encapsulation of the detached piece. We have dealt also with the overgrowth of fat in connection with diet, and perhaps this would be the best place for me to speak of the general influence of diet in respect to obesity. Of course, we have far more examples of general obesity—an increase of fat in all parts of the body, not equally in all parts, but still a general increase, and not merely locally, as in the cases just described. We get ordinary obesity in connection frequently with diet, but not altogether. In a great many cases, and in, I believe, the extremest instances of obesity, it would not appear that diet has much action. In the lesser cases, which show a little deviation from the ordinary standard, if you were to question the patients you would find beer-drinking habits in connection with consumption of large amounts of fat and saccharine matters, but in others not so. The next point is with regard to race. Obesity of the extreme sort is very common with Zulus; Lobengula had this condition himself. He was enormously fat, and a great many of the Kaffir men are extremely fat. In them one often meets with pendulous breasts in the male, not due altogether or chiefly to hypertrophy of the mammary glandular tissue, but to the increase of fat around the mammary gland. As we all know, the region of the mammary gland, both in the male and in the female, is one in which there is apt to be an accumulation of fat. Under such circumstances the male breast may take on very much the appearance of a female breast. But if you cut such a breast across post mortem, I suspect that you will find exceedingly little mammary glandular tissue. As age advances women who have any

tendency to fatten will generally show it in the region of the mammary gland, but it will be found that that is not due to any hypertrophy of the gland itself, but to the enormous amount of fat which has been deposited there. That is seen in the case of a scirrhus breast, in which there is a nodule of scirrhus, and there is only very little of the mammary gland, though there is surrounding it a great pendulous mass of fat. I have seen more than one breast which was removed for cancer in which I could scarcely find any mammary gland at all: there was just the tumour and the mass of fat. The gland had been absorbed, and the fat had taken its place. Before the operation it looked as if we had to deal with an enormous gland.

I have said that Kaffirs are very liable to get fat. That is the case with negroes too, sometimes. That race tendency, however, is generally found to have some connection with beer. Kaffirs drink their own beer, and it is of a very fattening description; moreover it contains a great deal more unfermented substances than we should fancy or than our own beer contains. I have said that negroes show the same tendency to fatness, and they are consumers of large quantities of sugar. There are some races of people who do not fatten, just as there are some animals which do not. You all know that a pig can be easily made to put on fat, and so with other animals in various degrees. It is exceedingly difficult to fatten a goat. A goat may be fed very liberally, but it will never look as if it had enough to eat.

I have alluded to the influence of diet, and the influence of race, and now I may mention family tendencies. Some families tend to be fat. In South Africa it would appear that most of the races have some tendency to fatten. The Boers fatten, and the Boer women do so very much indeed, so that you can scarcely meet a Boer woman who has not grown very fat; and it is also asserted that Boer women almost invariably lose their front teeth at thirty or thirty-five years of age. I do not know how true that statement is. Men do not fatten so readily or to such a degree, and they preserve the teeth. Whether there is any connection between the occurrences I do not know, but it is only a rough observation. But there is no doubt it is a general tendency, and that the Boer women fatten there can be no question whatever.

Now I have to make one other suggestion, which takes us into another department of physiology, in reference to the tendency to accumulate fat, and that is its relation to the sexual system. It is well known to cattle dealers that removal of the ovaries or the testes from an animal which has been allowed to retain them to adult life results in the animals showing a decided tendency to put on fat. If a man wishes particularly to fatten a sow he will have her ovaries removed. It is not necessary, but it is considered to very definitely influence the putting on of fat. Conversely, the physiological use of the sexual system and the liability to sexual excitement has a tendency to leanness. Very few persons with highly developed sexual habits will put on fat to any large extent. A suppression of the sexual system and function may account for the most definite tendency to fatten about the mammary region. This occurs not infrequently in quite young persons; young women may get enormously fat, and in such cases you will very probably find that the sexual system is in abeyance. In the case of the male you will find that the testes are very small, and that he has practically no development of sexual hair. That constitutes a definite class of cases. I will not go further than to say I think there is a definite relation between the sexual functions and the tendency to fatten, and that there is a definite group of cases, occurring chiefly in young men, in which the sexual system remains in abeyance; the voice is weak, and the patient is enormously fat and has big mammary glands, which are probably chiefly fat. I have seen some very remarkable instances of that, and they are not usually attended with high intellectual development; I mean that the patient who has the sexual system partially suppressed and an abnormal growth of fat is more or less wanting in mental energy.

We have curious cases in the female sex of the same thing. In men, so far as I have seen, the cases to which I have just referred begin in quite early life, and the tendency to excessive increase of fat is from boyhood, and the testes are extremely small. That will only occur quite in early life. But in women the sexual function may be suppressed, and we may have what we may call almost an acute tendency to lipomatosis. A girl who has been perfectly healthy and who has menstruated well and regularly, and enjoyed good health, may

suddenly have a complete suspension of the menstrual function, and may put on fat to an enormous extent. Of this I have in my mind at the present time one very remarkable case, that of a young lady who was nearly six feet in height, and who, I heard, enjoyed excellent health until sixteen years of age. She was eighteen years of age when I saw her. At sixteen, menstruation, which had been well established, was entirely suspended, and she at once began to fatten to an enormous extent.

There is one little symptom which I should mention in connection with the tendency to develop fat, and that is the tendency to develop scars in the skin, that is to say subcutaneous scars. Here we have a reference to the scars in the bark of trees. The expansion and growth of the tree is in excess of the power of the bark to yield, and the bark gets torn, not openly in the surface, but in the deeper parts. Sometimes a slash is made in the bark to allow it to expand. The growth of the skin is generally *pari passu* with the growth of the fat; but in these cases of abnormal development of fat it is not so. Everyone knows the scars which are to be seen on the abdomens of mothers, and the scars caused by the excessive increase of fat are like those. The cases in which a slight tendency to fatten on certain parts of the body is present are infinitely more common than extreme examples of fatness; and those who are accustomed to see patients stripped find some who have never been out of health, but in whom these little scars occur over the hips and shoulders and in various parts of the body, showing that there has been at some time a tendency to fatten more than the skin would conveniently allow.

To return to the young lady whom I have already referred to, in whom menstruation had stopped, and in whom there was a sudden tendency to put on fat in all parts of the body; she was dotted all over with these scars, which developed very quickly over the shoulders and the arms, the skin not yielding to the growth of fat beneath it. She was extremely disfigured by these scars. I will just anticipate what I have to say about the treatment by telling you that in the case of this young lady the use of extract of ovary appeared to do a great deal of good. Many medical remedies were tried, and although I must not attribute the benefit to any one remedy (the growth of fat was enormous,

and at the same time she was very weak and lethargic), yet under treatment by the extract of ovary she has regained her health to a very large extent, and menstruation has recurred. There is a possible fallacy in attributing the cure to any one thing; but at any rate her tendency to manufacture fat in abnormal quantity has ceased, and she now appears to be in no danger of death from this, which at one time was feared.

I believe that all these cases of great tendency to fatten must be regarded as unsatisfactory in reference to prognosis. I think a very fat person scarcely ever lives to a real old age. These people nearly always take on some form of debility. One risk is this: that the heart may become embarrassed. Although fatty degeneration of the muscular substance of the heart and the accumulation of fat around the heart are two distinct things and must be kept separate, the accumulation of fat about the heart is not to be wholly disregarded. In lipomatosis there is fat about the heart, and the heart is more or less enfeebled. That is explained by some by the formation of a strong adhesion between the muscle of the heart and the fat on the other side; but modern pathologists have a tendency to go back on the old-fashioned idea and to think that there is some association, and that fat does accumulate amongst the muscles. Fatty degeneration of the heart has no tendency to cause the accumulation of adipose tissue, but in the patient who has heart disease, the tendency to the accumulation of fat is a source of danger in after life.

Other sources of danger attend extreme cases of obesity. You will find in many books lists of cases of enormous obesity, such as Daniel Lambert; and in a book recently published in America, by Dr. Booth, there is a long list of these cases of enormous increase of fatty tissue. In some death has been quite accidental owing to inability to move; and the quantity of fat has embarrassed the movements of respiration. One poor woman was so fat that having turned over on to her face in bed was quite unable to turn back again, and was therefore suffocated. Others have been very nearly suffocated. Some when seeking to regain the erect position have died from embarrassment owing to pressure on the trachea. As an example of a rather rough means of preventing accidents, a surgeon had needles put into the back of the

patient's chair so as to prevent him leaning back and going to sleep and thus being suffocated.

Next I will say a word as to the occurrence of congenital lipomatosis. Such cases are the infants who are sometimes exhibited in shows. In these cases one seems to have no very clear explanation to give. It is very seldom hereditary, though now and then two members of a family are affected with it. Generally it occurs in only one member of the family, and it may be manifested within a very short period after birth, showing that there was a very distinct tendency to it irrespective of any condition supervening afterwards. The food may be of the ordinary kind and of the usual amount, but yet the child puts on fat with extraordinary rapidity, although there is no very great increase in appetite. That is also the fact in many cases in the adult where there is over-fattening—the consumption of food is not very large. In these children who quickly become fat there is sometimes a tendency to precocious development in other respects. I believe in many cases where there is this early tendency to the development of fat that the sexual function is suppressed, or nearly suppressed. But this is not the case in all, and not so much in the female sex as in the male. Young girls tending to be fat may at eight or nine years of age have all the appearance of a girl of fifteen or sixteen; very often they are precociously developed in other matters. These fat children, I suspect, never live long; I do not know of any recorded case in which they have lived very long; they are feeble, and if they grow up to adult age they never live far beyond it. Cases of congenital corpulency vary, and we must admit that they are inexplicable. They range themselves into a tendency to overgrowth of some other parts, and in some there is a tendency to dwarfing.

We have recently got a little further in the explanation of the tendency to giant growth in general in connection with the pituitary body. That has some power of regulating the growth, just as it produces symptoms in the disease known as acromegaly. In that disease the pituitary body is larger than normal. So I think we shall find that some part of the structure presides over the tendency to fattening. I might allude to the fact that some have connected this increased fatness with the thyroid body, and they used the extract of thyroid to prevent the accumulation of fat. I

do not think that is so. If you give thyroid extract certainly the patient loses flesh and strength, and is very weak, but in the case of the young lady I have mentioned who had an enormous tendency to fatty formation the thyroid extract quite failed to prevent it.

As regards the weight which may be attained by these people, it is sometimes enormous. There is an instance on record of one of these congenital cases of corpulency which by the time it was fourteen or fifteen years of age was so enormously fat that it was decided to take the child for exhibition, but it could not be got out of the house; it had lived in one room for so long and had attained such proportions that it could not be got out of the door. The enormous weight of sixty stones is recorded in a case of general diffuse lipomatosis, and that was in a negress. I believe that is the greatest weight on record. Daniel Lambert, with whose name we are all familiar as an example of extreme development of fat, scaled eight stones short of that, namely fifty-two stones. In not a few cases weights very nearly approaching to these have been attained by fat persons. In most cases a patient suffering from this diffuse tendency to enormous accumulation of fat becomes lethargic, and there is a great tendency to sleep; but in some instances they maintain a fair amount of mental activity. In some cases there are no aberrations, but a general condition of willingness to remain half asleep during the waking period. Daniel Lambert's intelligence was very fair, and he maintained for the greater part of his life a considerable amount of cheerfulness.

Quite recently a new affection has been described under the term lipomatosis dolorosa. In the ordinary diffuse lipomatosis there is no pain with the fatness, but a physician in Philadelphia has described cases under this name, and the feature is that the patient complains very much of aching in the affected parts. But the amount of lipomatosis in these patients does not approach that in the ordinary diffuse form. It is often local, it affects the breasts and parts of the abdomen, which may become pendulous. We will therefore keep in mind the fact that it has been observed that there is a tendency in the adult to put on a large quantity of fat, especially about the mammary glandular region, and the lower part of the abdomen, and the lower parts of the arms, and that it

has been described as attended by pain, and though it is a different condition, it is probably related in some way to myxœdema, in which disease the cellular tissue increases and becomes charged with *pseudo*-hypertrophy in many parts. In the other case you have a great increase of fat, and you distinguish it from cases of myxœdema by such symptoms as absence of the spade-like hand, and the fact that there is not a loss of hair, nor is there present the peculiar facial expression of myxœdema. I have had no opportunity myself of observing any case of this so-called painful form of lipomatosis; but we will keep it in mind in case anything occurs in connection with it.

Before I leave the subject of fatness, I should like to mention one example of the enormous fatness occurring in early life. It was in a woman who had no sort of arrest of sexual function. She was married, and was the mother of several children; but that is exceptional. You will find, as a rule, that very fat women have not been parents of children, and there is every reason to believe that in them the sexual functions are in abeyance.

Now just a word as to the converse picture, that is to say, living skeletons, those in whom the subcutaneous fat is remarkably absent, and the conditions under which this may occur. This happens to patients in various conditions of health. It is more common in women than in men. Extreme emaciation may occur and remove the whole of the subcutaneous fat, and very often that happens in association with some loss of sexual function. There is often an absence of menstruation in a young woman who becomes abnormally lean; but the condition of leanness may occur coincidentally with very fair menstruation, and with quite good health.

It has been noticed that a person who has enjoyed the ordinary development of subcutaneous fat up to middle life may suddenly become very lean indeed. There are some remarkable examples of that which have occurred without any noticeable departure from general health. To get lean as one gets old is, I believe, rather a good sign if it is not extreme. To lose fat as you get old, or at all events not to gain it, is a happy augury as regards long life. But some of the extreme instances of living skeletons are examples of disease; some of them are the subjects of nervous disease, or of

disease of the spinal cord. Others, again, arise from some ill-understood cause. In connection with these living skeletons, we must mention a few cases which fit in with the cases of congenital lipomatosis, in which the infant becomes very fat. We see infants which are very lean, but that is a very much rarer condition in infants than for them to become fat. Of this leanness I have seen two remarkable examples; they were both attended with dwarfing, that is to say, the patient did not grow well. In both the sexual system remained quite in abeyance, the testes being quite small, although the patients had attained nearly the usual age for development of function. One of them is dead, but the other is still living. In both of them the tendency to leanness was so great that you could not appreciate any subcutaneous fat in any part of the body, and you might have thought there was no subcutaneous and cellular tissue, as the skin could be felt close on to the muscles and the bones, and the contour of the muscles could all be seen. The muscles themselves were in very fair condition. In both of them the voice remained infantile, and the testes were only little lumps. Both of them were boys, and in one of them the mammary glands and the nipples were quite absent. There was a very peculiar hereditary history. In both the hair was absent; that is to say, the appendages of the skin as well as the subcutaneous fat were absent almost wholly, and the scalp was tight over the whole of the skull.

A very peculiar fact, which I do not like to mention as if I thought it had any real bearing on the case, is that the mother of one of the boys was quite hairless herself. She was a finely-developed lady with plenty of fat and large glands, but she had no hair on her head. But she was not born hairless, she lost her hair in childhood. Under what laws of hereditary tendency was there produced in her offspring this remarkable absence of hairy appendages? Apparently the glands of the skin were absent; it was dry, and no sebaceous glands seemed to be in operation. This lady had a family of several daughters, four or five, and this was her only son. Her daughters all had excellent heads of hair, and were well developed in every way; but the only son of this hairless woman was born entirely hairless, without mammary glands or subcutaneous tissue. In the case which died a post-mortem was obtained, but nothing which

could furnish any clue to a cause was found. The clavicle was very small and slender, like that of a little animal, and not so thick as a cedar pencil. Those are two cases which illustrate the condition, and they are so exactly alike that I constantly mistake one photograph for the other, and the two show that there must be some definite cause for such arrest of development. There is nothing to be suggested as regards any previous deformity or defect in the parents.

That concludes what I have to say about excessive development of fat, and its deficiency.

As regards treatment, there is very little to say. I have said in passing almost all I have to say on that matter. It is thought that the use of alkalies is good. Our grandfathers used to give liquor potassæ, but we have lost confidence very much in that. I have no doubt that the absence of fatty material is of extreme importance, and if we feed the patient on beef-steaks and hot water in order to eliminate fat it is very efficacious, and many schemes of dietary for corpulency have been devised. They go with the cases of general functional corpulency, which are more or less amenable to dietetic treatment. In these more definite cases of great excess there is apparently little to do in diet, but you should carry out the same methods in them to avoid those foods which can be easily changed into fat. Beyond that I do not know of anything special to be recommended. Thyroid extract could be tried, but cautiously, because by its administration you will very likely reduce the patient's strength out of proportion to the beneficial effects.

With regard to surgery in those conditions, I have nothing to say beyond what I have already said with regard to the removal of isolated fatty tumours, and the removal with caution of the more inconvenient tumours.

WE have received from Burroughs, Wellcome & Co., a specimen tabloid of Hydrarg. perchlor. gr. $\frac{1}{8}$ et Pot. iod. gr. v. This preparation will be found very convenient in practice, and it will be useful to bear in mind that the gr. $\frac{1}{8}$ of the perchloride of mercury is equivalent to one drachm of the liquor hydrarg. perchlor. In those cases where the continued administration of iodide of potassium and mercury is desirable, this tabloid will be found serviceable and effective.

CLINICAL LECTURE

ON

"SOME CASES OF OVARIOTOMY."

Delivered July 5th, 1901, at the Royal Free Hospital.

By JAMES BERRY, B.S., F.R.C.S.

At the present time there are, in my female ward, three women from whom ovarian tumours have recently been removed. As each of these cases presents some points of interest, they may well form the subject of a clinical lecture upon the diagnosis and treatment of ovarian tumours. I will take the opportunity also of mentioning briefly some of the other ovarian cases that have from time to time come under my care. I intend to deal with the subject entirely from the point of view of my own personal experience.

CASE 1. *Phthisis; ascites; ovarian tumour; ovariectomy*.—Gladys M—, a delicate-looking single woman, æt. 20, was admitted on June 14th, with the following history:—In October, 1900, she first noticed a swelling in the lower part of the abdomen; this has gradually been increasing in size. In December her menstruation ceased, and it has not returned. In January, 1901, she was laid up for a week with pulmonary symptoms, and it was then discovered that she was suffering from phthisis. About this time the abdominal swelling became somewhat smaller, but it subsequently continued to increase in size.

In March she was admitted to the North London Hospital for Consumption, where the diagnosis of pulmonary phthisis was confirmed. While under treatment there the abdominal tumour was noticed and thought to be ovarian. A few weeks later it was noticed that the abdomen contained a large quantity of free fluid, and as her phthisis had in the meantime considerably improved, she was sent to the Royal Free Hospital for the treatment of her abdominal trouble. Those of you who saw her on admission will have noticed that the abdomen was uniformly and greatly distended. The shape was, however, by no means characteristic of an ovarian tumour, for the abdomen was markedly flattened in front and bulging in the flanks. This shape of abdomen naturally suggested ascites, and on percussion this diagnosis was confirmed by finding that there was dullness in both flanks and in the hypogastrium, the only

resonant area being in the umbilical and epigastric regions. When the patient was turned on her side the area of dullness shifted, and the side that was uppermost became resonant, while the rest of the abdomen was dull. The diagnosis of a large quantity of fluid free in the peritoneal cavity was therefore clearly established. But on palpating the abdomen it became evident that there was more than this. In deep palpation somewhat below and to the left of the umbilicus a rounded mass could be felt, apparently about the size of a foetal head. This mass was best felt by suddenly depressing the fingers upon it, when a sense of ballottement was obtained. The tumour appeared to bob about in the fluid in which it was floating. It moved about freely in the lower part of the abdomen, but could not be displaced above the level of the umbilicus. It was evidently anchored to something at the lower part of the abdomen. The large quantity of fluid in which it was floating prevented its physical characters from being easily ascertained, but it appeared to be round, smooth, and fairly hard. In short, it appeared to be an ovarian tumour. But before jumping hurriedly to this conclusion several other points had to be considered. Of these the most important were:—(1) Could this lump be anything else but ovarian? (2) What was the cause of the ascites?

1.—The free mobility of the tumour from side to side, and the impossibility of displacing it upwards, pointed strongly to its having a comparatively slender attachment to some pelvic organ, probably within the ovary or the uterus. The question of pregnancy suggested itself, more especially as there was a clear history of six months amenorrhœa. It is true that the girl was unmarried, but then it should be remembered that it is precisely in unmarried girls that the mistake of taking a pregnant uterus to be an ovarian cyst is most often made. The examination of the breasts showed these to be quite small, and quite unlike the breasts of advanced pregnancy. Examination of the vagina was somewhat difficult, but the cervix uteri could just be felt, and was not softened. The uterus examined *per rectum* was found to be pushed downwards and forwards, and movement of the tumour did not appear to influence that of the uterus. A uterine sound, which was, however, not passed until it was certain that pregnancy did not exist, showed a

uterus of normal size, unconnected with the tumour.

The question of uterine fibroid was easily answered in the negative; the age of the patient, the free mobility of the tumour apart from the uterus, the downward displacement of that organ, the complete absence of menorrhagia or metrorrhagia, and the result of examination by the uterine sound were, taken all together, enough to exclude the possibility of our having to deal with any kind of uterine tumour. It was suggested that perhaps the tumour might be connected with the intestine, that it was a mass of small intestines matted together by old inflammation, with or without the omentum. Such masses are not very uncommon in cases of tuberculous peritonitis, and in this case one could not ignore the fact that the patient was actually suffering from tubercle of the lungs. The smoothness and roundness of the lump, and the absence of any similar lumps in other parts of the abdomen, together with its low situation and its obvious connection with the pelvis, made such a diagnosis in the highest degree improbable.

Now that I am on the subject of tuberculous peritonitis I may mention that occasionally a localised collection of peritoneal fluid may very closely simulate an ovarian tumour. Some years ago, when surgical registrar at another hospital, I had to make a *post-mortem* examination on a case that illustrated this point very clearly. A young woman had been admitted acutely ill, with a large tender abdominal swelling. The abdomen was found to contain a rounded prominent swelling, situated exactly in the middle line, and about as large as a man's head. There was resonance in both flanks and in the epigastrium. The diagnosis of ovarian tumour was made, but when the abdomen came to be opened it was found that both ovaries were normal, and that the supposed ovarian tumour was merely a large collection of peritoneal fluid due to tuberculous peritonitis. The fluid was limited above by the transverse mesocolon, and on either side by omentum and intestines, adherent to the anterior abdominal wall. The great distension of the abdomen had probably prevented the surgeon from feeling the inflammatory bands and lumps which were present in other parts of the abdomen. The signs of bronchitis which were present in the lungs were due to

tuberculosis, and might have given a clue to the diagnosis, but were attributed to the causes already mentioned.

2. I turn now to the question of the cause of the ascites in our case. There was no general anasarca such as might be caused by renal or cardiac disease. There was no evident disease of the liver. It seemed clear that the ascites was due to one or other of two causes, either tubercle of the peritoneum or the ovarian tumour. In favour of the former was the fact that the patient was known to be suffering from tubercle of the lung. On the other hand, nowhere in the abdomen could any evidence be found of those inflammatory masses or bands so characteristic of that disease. Careful palpation did not reveal that transverse ill-defined band so often felt in such cases in the neighbourhood of the umbilicus, and which is due to the thickening and matting together of the different parts of the great omentum. In short, there was no evidence of tuberculous peritonitis. Nevertheless, tuberculous peritonitis may present itself in the form of a simple chronic effusion, and the co-existence of ovarian tumour with tuberculous peritonitis, although rare, is by no means unknown. Some of you may have seen that a case of this unusual coincidence was published by Mr. A. C. Roper, of Exeter, in the *Lancet*, on the very day on which our patient came into the hospital.

If the ascites then was not due to tubercle it was almost certainly due to the ovarian cyst. I need hardly say that ascites is a very common accompaniment of ovarian tumour. Sometimes it is due to malignancy, sometimes to inflammation or strangulation of the tumour, sometimes to rupture of the cyst or of some portion of it.

In this case I did not commit myself to any definite diagnosis as to the cause of the ascites, although feeling pretty sure that the tumour was ovarian; and being rather inclined towards the belief that the peritoneal fluid would prove to be in some way connected with tubercle, it seemed best to leave the diagnosis somewhat uncertain until after operation; for it was quite clear that whatever the nature of the fluid, there was only one form of treatment that could be adopted, namely, abdominal section, removal of the fluid and of the ovarian tumour. When the abdomen was opened it was at once evident from the viscid nature of the fluid that it had an ovarian origin. After removal

of the tumour, which proved to be a multilocular ovarian cyst with considerable solid growth, a ragged aperture was discovered in one of the cysts, and it was clear that on some previous occasion the cyst had ruptured into the peritoneal cavity. The extravasated fluid had caused a slight degree of peritonitis, shown by the general injection and slight thickening of the parietal peritoneum. This slight peritonitis had led in its turn to further effusion, which accounted for the very large amount of fluid that was free in the abdomen. The operation presented no difficulty of any kind, and there is nothing to be said about the subsequent progress of the case, which was quite normal and uneventful, as after a simple ovariectomy it always should be.

CASE 2. *Large multilocular ovarian cystoma with slight peritonitis; ovariectomy.*—Emma D—, a married woman, æt. 34, was sent into the hospital on June 22nd, on account of a large abdominal swelling. The chief points about the history were that for twelve months the abdomen had been gradually increasing in size; her menstruation had never been very regular, but in the last nine months, according to her own account, she had menstruated about once a fortnight.

In spite of this she had believed herself to be pregnant, and had engaged a doctor to attend her. It was not until the time of her confinement was, as she thought, long overdue, that further investigations were made by her doctor, as the result of which he sent her to the hospital.

The patient was a small, thin woman, and her abdomen presented a striking contrast to that of the previous case. It was greatly distended, measuring $37\frac{1}{4}$ inches in circumference, and very prominent; there was no bulging of either flank.

Resonance was marked in the flanks and epigastric regions, and absent from all other parts. It was obvious that the swelling was due to a large tumour which practically filled the abdomen. The tumour was smooth, tense, and somewhat tender; a fluid thrill was easily obtained.

When the patient was asked to sit up, the contraction of the abdominal muscles in front of the tumour could be easily seen and felt. When she took a deep breath, a coarse friction could be felt by a hand placed upon the upper part of the abdomen. This indicated a roughness of the opposed visceral and parietal layers of peritoneum, and was seen at the subsequent operation to be

due to a localised peritonitis. The slight movement of the tumour and abdominal wall upon each other appeared to indicate the probable absence of anterior adhesions or, at any rate, of extensive adhesions. There was no free fluid in the peritoneal cavity.

The uterus, which was pushed downwards by the tumour, had the characters of the unimpregnated organ, and the condition of the breasts also negatived any idea of pregnancy. The existence of any uterine tumour was easily excluded. The diagnosis of an ovarian cystoma was easy, and admitted of no doubt. In all other respects the patient appeared to be healthy.

Ovariectomy was performed in the usual manner and without any difficulty. The tumour was a multilocular cystoma, one of the cysts being far larger than all the others put together.

After the tumour had been removed, the opposite ovary was drawn up and examined. It was found to contain two or three small cysts not larger than peas. It was not removed as it was not by any means certain that these cysts would ever cause trouble, and the objections to removing both ovaries are so serious that this proceeding should be avoided unless absolutely necessary.

This patient, like the first, is making an excellent and uneventful recovery.

CASE 3. *Large dermoid cysts of both ovaries; twisting of pedicle, causing strangulation of one of the cysts; ovariectomy.*—This case differed considerably from the two preceding, and is, in some respects, a very remarkable one.

Hannah McT—, a widow, æt. 61, who had had one child, now aged about thirty-five, was admitted on June 27th, on account of high temperature and abdominal pain and swelling.

Thirty years ago she first noticed a lump in the lower abdomen, which caused her little or no trouble, and to which she paid but little attention. Seventeen years ago she consulted a doctor, who told her she had "three ovarian cysts." This statement I was at first inclined to disbelieve, but as you will hear, there was more truth in it than I suspected. No operation was performed at that time.

Seven years ago she had a bad attack of abdominal pain, and had to keep to her bed for some days; she appears to have been acutely ill at this time. She recovered from this, however, and appears to

have suffered little trouble afterwards, except from the size and weight of the tumour or tumours, until June 23rd of this year, when she was again seized with abdominal pain and nausea. This pain continued to be severe, and her temperature varied from 100° to 102°. Her bowels were not opened in spite of the fact that she had dosed herself with aperient medicine. Vomiting was not a prominent feature, but she had constant nausea and was unable to keep any food down. Her own medical attendant, Dr. Lewis Hawkes, who was then called in, recognised that she was acutely ill, and asked me to see her with him.

With some difficulty we persuaded her to go to the hospital. She was, perhaps not unnaturally, much averse to any operation, as she had been advised by others years ago, when ovariectomy was a much more serious proceeding than it is now, to have nothing done. It was the acuteness of her symptoms, however, and the fact that she was evidently getting worse that made us urge the operation upon her, and it was well that we did so. On admission, many of you saw that she was a thin frail woman with white hair, looking a good deal more than her actual age (sixty-one). The abdomen was occupied by a large tumour extending upwards beyond the umbilicus. It was irregular in shape and extremely hard. It was but very slightly moveable from side to side, and was evidently fixed to some pelvic organ. It was acutely tender, so that examination was not very readily permitted. Its extreme hardness and irregularity, together with the long history, at first suggested a fibroid of the uterus. But the menstrual history was that she had never had menorrhagia or metrorrhagia at any time, and that the menopause had occurred nine years ago. It is true that old subperitoneal fibroids, especially those with slender pedicles, may be unaccompanied by uterine hæmorrhage, but it is unusual to find that there never has been such hæmorrhage at any time.

The vaginal examination confirmed the idea that we were not dealing with a fibroid. It showed much more than this, however. The uterus was small, and pushed downwards and forwards, so that it was closely applied to the back of the pubic bones. Douglas's pouch was occupied by a large, smooth, elastic swelling, which occupied the whole of the pelvis and was quite immoveable. This swelling was evidently a cyst with a fairly thin wall,

and it had apparently no connection with the abdominal tumour.

The diagnosis made was that of at least two ovarian cysts, one of which was small and impacted in the pelvis, the other large, hard, and inflamed. Dermoid cyst was thought of, but not positively diagnosed, as, perhaps, it ought to have been. It was thought, moreover, that the inflammation of the abdominal tumour was probably due to twisting of the pedicle, although the possibility of the inflammation having its origin in some neighbouring part could not be wholly excluded.

The urgency of the case made it undesirable to wait until my ordinary operating day came round.

On June 28th the abdomen was opened, and a remarkable state of affairs disclosed. The parietal peritoneum opposite the tumour was greatly thickened from long-continued friction against the tumour. It formed a dense, tough, fibrous plate at least a sixth of an inch in thickness, and cut like cartilage. You will observe a similar thick plate of fibrous tissue on the peritoneum covering the tumour, which I show you now.

The larger tumour was at once seen to be a huge dermoid. At the upper part was the omentum, extensively adherent, and forming a thick pedicle. This had been twisted axially no less than four times, and its distal end was swollen, and of a purple colour from strangulation. After ligature and division of this the tumour was lifted out of the abdomen. It was then seen that the Fallopian tube and neighbouring part of the broad ligament were also greatly swollen and thickened, forming a mass as large as two fingers, and also of a deep purple colour. The most curious point about the case, however, was that the original pedicle, by which these structures were attached to the pelvis, had been twisted so many times, evidently years ago, that the blood supply in it had been completely, or almost completely, cut off. It had been reduced to a mere fibrous cord about an eighth of an inch in diameter, or about as thick as a piece of stout whiplcord.

It seemed clear from the amount of atrophy that had occurred that this twisting of the pedicle had taken place years before, probably at the time of the acute illness seven years ago, to which allusion has already been made.

The vitality of the tumour had been maintained during these years through the omental pedicle,

and it was when this, in turn, became twisted that the recent urgent symptoms set in.

The larger tumour having been removed, the pelvis was investigated. With difficulty I could just get my fingers into the pelvis by the side of the second tumour, which completely filled it. After removal of this pelvic mass, which, fortunately, was not adherent anywhere except by its fairly wide pedicle, it was found to consist of two distinct tumours, loosely united by a slender band. One was a thin-walled dermoid cyst nearly as large as a foetal head, the other a very thin-walled, translucent cyst about as large as a duck's egg, evidently a parovarian cyst. So the original diagnosis of "three ovarian tumours" was after all proved to be correct.

That the two larger cysts were dermoid was proved not merely by their yellow colour, but by their contents. Both contained a large quantity of viscid sebaceous fluid looking like cream, as well as a large ball of hair. The larger one had in its wall three pieces of jaw-bone, in each of which were two or more well-formed teeth; the smaller cyst also contained teeth and a piece of bone. After the operation the temperature dropped at once to the normal, and remained there. The patient lost all her pain, and is now making an excellent recovery, having had no bad symptoms of any kind.

Hair, bones, teeth, and sebaceous matter are, of course, characteristic constituents of ovarian dermoids. Here is another specimen of considerable size containing two pieces of jaw-bone with teeth, that I removed two years ago, on account of recent inflammation, from a lady æt. about 30, who had not been aware of the existence of any tumour until a few days before I saw her. Dermoid tumours, as a rule, increase in size very slowly, and differ in this respect from most ovarian cysts. Often the long duration of the tumour affords a clue to the diagnosis of an ovarian dermoid.

Here is a third specimen of an ovarian dermoid of considerable size, and weighing four pounds, that I removed in this hospital last year from a woman æt. 47. She had been aware of its presence for several years. It contained a quantity of thin sebaceous matter, a small ball of hair, and several teeth.

(To be concluded.)

MYASTHENIA GASTRICA: ITS DIAGNOSIS AND TREATMENT.

By GEORGE HERSCHELL, M.D.

(Concluded from p. 159.)

Myasthenia with Retention.—The stomach is now absolutely unable ever to completely empty itself of its contents, and always contains a fermenting residue. The stomach is ever in a septic condition, and food will commence to ferment almost as soon as it is introduced. This condition of inability to empty itself in some cases commences suddenly, as the result of imprudence in food, from over-fatigue, or influenza, or any depressing cause which may supply the proverbial last straw. I have seen it induced apparently in a reflex manner from the passage of a gall-stone. In most cases the change from myasthenia without, to myasthenia with, retention is more or less gradual. The patient will have, at intervals, a succession of attacks, during which the stomach does not completely empty itself. Vomiting occurs, the patient is put upon a low and restricted diet for a few days, and the stomach recovers itself for the time and resumes its *status quo* of myasthenia of the second degree, in which it is able to empty itself completely during the night. These attacks become more frequent until the condition is firmly established.

In addition to the symptoms already described, which will increase in intensity, the patient will suffer from others due to the following factors:

1. The quantity of water which leaves the stomach and passes into the duodenum is much reduced. From this naturally follows extreme thirst and constipation, with diminution in the quantity of urine.

2. In the first and second stages of myasthenia, fermentation does not take place in the stomach to any serious extent. The chyme which passes from the stomach into the duodenum still contains nutritious substances, and can be utilised for further digestion in the intestine. It is far different in retention myasthenia when well established. Not only is the peptonisation in the stomach reduced to a minimum, but the stomach contents which actually reach the duodenum are disorganised, and are neither useful for nutrition nor for further digestive processes. We shall then have as symptoms:

(a) Emaciation of the patient from actual starvation.

(b) Various intestinal troubles set up from the irritation of the putrefying mass which is introduced into it through the pylorus.

There is certain to be intestinal flatulence, and often localised pain. Diarrhoea is sometimes met with from the irritation of the intestinal contents and the fermentative processes going on in them, but more often we find constipation produced partly by the deficiency of water which reaches the intestines, and partly by localised spasm of the large intestine. The condition of the patient is, in this latter case, much graver, as the intestines not being able to empty themselves of their poisonous contents, toxins are absorbed into the system and neurasthenia of a severe type is produced. Among the several intestinal troubles muco-membranous colitis especially is often met with, and is a very distressing complication.

(c) The irritation of the fermenting food in the stomach will produce pain and nausea. As with successive meals the residual material in the stomach increases, a point will at last be reached at which vomiting will occur. This will happen in well-established cases about every fourth or fifth day.

The most important feature, especially with regard to prognosis, is the starvation of the patient. No food which is taken is utilised for purposes of nutrition except that consumed during the first few hours immediately following an attack of vomiting.

The objective signs of myasthenia with retention are characteristic, and are as follows:

1. There will be splashing in the stomach before breakfast.
2. There will be food residues in the stomach before breakfast.

The chief affection from which we shall have to differentiate this stage of myasthenia is obstruction of the pylorus, either benign or malignant. The chief points of difference are that peristaltic movements of the stomach will not be visible in myasthenia, and by the water test already described we shall see that the stomach has a great difficulty in dealing with liquids. In obstruction, on the other hand, unless advanced to that stage when its diagnosis by other means would be obvious, the converse is the case. We can also learn a great deal from the behaviour of the stomach during lavage.

In cases of obstruction, the liquid which has been introduced into the stomach can be removed by making the patient strain as in the act of coughing or defæcation. In fact the liquid will often spout out of the end of the stomach tube before the operator has had time to attach the tube leading to the exhausting apparatus. In myasthenia, on the other hand, the first thing which will impress a medical man who is attempting to wash out the stomach will be the extreme difficulty of getting the water out which has once been introduced. Syphonage or aspiration are always necessary.

Again, in pyloric obstruction, splashing, if it can be produced at all, will only occur when there is a small quantity of fluid in the stomach, never when the stomach is quite full. In myasthenia of this stage the stomach will splash during the whole of the digestive period, and even directly after the stomach has been emptied as perfectly as possible by a tube. This latter fact, mentioned by Van Valsah, is probably due to the imperfect tubes in common use which, having two eyes at different levels, never permit the whole of the stomach contents to be drawn off.

Treatment of myasthenia with retention.—This is practically the same as in the non-retention forms with certain additions. The first point to attend to is to see that the patient's mouth is in an aseptic condition. It is futile and childish to expect to keep the stomach free from the germs of fermentation by any means whatever, as long as we are allowing them to be continually introduced from outside. We must therefore get a dental surgeon to remove all necrosed and septic stumps from the mouth, and cure any pyorrhœa alveolaris by the application of nitrate of silver or its equivalent, after all particles of tartar have been removed from the roots of the teeth.

The next thing to do is to see that the patient has an opportunity of absorbing into his system the daily amount of water which it requires. To this end we order an enema of tepid water to be given night and morning and retained. If a movement of the bowels should fortunately follow either of these, we at once administer another injection of a slightly less quantity which will probably be retained.

Having done these we may consider the question of washing out the stomach. On this subject much misconception exists in the profession.

Lavage is erroneously looked upon by many as a kind of panacea for all stomach troubles if the patient can only be induced to submit to it. In fact, such is far from being the case. By lavage we can do certain things, and we should never suggest it unless we have a clear conception in our own mind of the way we wish it to act in the case before us. In many gastric conditions wash-out the stomach would be the worst kind of treatment. In retention myasthenia, however, our course is plain. We must once a day empty the stomach of its foul contents, and if we do this before breakfast, we shall not be robbing it of any nutriment, as all that now remains in it is useless for the needs of the organism. We shall thus

action upon the stomach, and materially aid in restoring its muscular tone.

As in the non-retention forms of myasthenia we may spray the stomach out before meals with dilute hydrochloric acid and bitter infusions, or may give them by the mouth. We now come to the important question of diet. I am a strong advocate in this condition of giving only two meals a day—breakfast and a late dinner. If anything is taken in between it should be a soluble preparation of meat, such as carnigen. This is a whitish hygroscopic powder at once soluble in water and of a pleasant taste. Peptones I believe are irritating, and are not so useful. The main point to bear in mind is, that it is not so much what the patient eats as

FIG. 6.



give the stomach a fresh start and enable it to commence the day under happier auspices. The immediate effect will be to practically double the amount of food which is digested and absorbed into the system. Incidentally, the intestinal disturbances will cease because the gastric fermentations have been put an end to, and the condition of the patient will soon undergo a great change for the better. After the contents of the stomach have been removed, we may usefully finish the lavage with the intragastric needle bath (see Fig. 6).

This is much more efficacious than the ordinary lavage for cleaning up the stomach walls, and the forcible fine needles of hot and cold water which are used in alternation exercise a powerful tonic

the form in which it is presented to the stomach that is of importance. The patient may be allowed to select whatever tempts his appetite the most as long as it is taken in a finely-divided condition. Scraped meat, purée of vegetables, panada of chicken or veal, lean of mutton chops minced, pounded, and put through a wire sieve; all these are good. We must be guided by results. If we find that a reasonable proportion of any kind of food has passed out of the stomach we may safely allow it. If, on the other hand, we find an undue quantity of it in the stomach at the morning lavage we must reduce it in quantity and give it in a more finely-divided condition.

These, then, are the main points in the diagnosis and treatment of myasthenia of the stomach. I

am conscious that in the limits of the present paper I have been unable to give more than the merest outline of this important subject, but I hope that the little which I have said may stimulate the reader to study further this very common and distressing affection.

NOTE ON ENEMA RASHES.

By CHARLES BOLTON, M.D.,

Resident Medical Officer, University College Hospital.

THE fact that the administration of enemas is frequently followed by rashes has for a long time been recognised, and a very generally accepted explanation of their occurrence is that they are of toxic origin, the water of the enema dissolving certain substances from the feces, which are absorbed by the intestinal mucous membrane.

In a general hospital these rashes are of especial importance on account of their diagnosis from certain other rashes, with which they may be confounded, as (1) Infectious diseases—scarlet fever, German measles; (2) Drug rashes—mercury, belladonna, boracic acid; (3) Septic rashes; (4) Serum rashes—diphtheria antitoxin, antistreptococcic serum. It is therefore of great advantage to be acquainted with any facts which may throw light upon their prevention.

Having been informed by a nurse that in the hospital where she was trained soft soap was used for making the enemas, and that enema rashes were never seen, I determined to put this suggestion to the test, thinking that the *kind* of soap used might influence the occurrence of enema rashes, which might possibly be of the nature of drug rashes.

In January, 1901, I ordered soft soap to be used for making the enemas in half the medical and surgical wards of the hospital, and hard yellow soap in the remaining half of the wards. This order continued in force for six months.

During this period a total of 903 enemas was given to 500 patients, of these 407 soft soap enemas being given to 234 patients, and 496 hard soap enemas being given to 266 patients. In the latter series seventeen rashes resulted, but in the former series I did not find a single rash. Enemas of pure water were not used, as it was considered

that if soft soap enemas did not cause a rash, neither would water alone.

Three hundred of the patients received only one enema each, the remaining 200 having from two to six each, and a few of the more chronic patients from twelve to fourteen each.

Of the seventeen rashes never more than one occurred in the same person, and when more than one enema was given the rash followed the first, second, third, or fourth, but not the later ones. In one case, after a patient had received several soft soap enemas the mistake of giving him a hard soap enema was made, and a rash resulted. Calculating from the number of hard soap enemas employed, the percentage of rashes is about $3\frac{1}{2}$. If the percentage of rashes be calculated from the number of patients who received these enemas, it amounts to about 6. These percentages, of course, are of relatively little value on account of the extremely limited nature of this investigation.

The medical and surgical diseases from which the patients were suffering had apparently no influence on the rashes, and the same remark may be made concerning the aperient employed. This was usually given the night before the administration of the enema, and consisted either of calomel, castor oil, or house medicine.

All the rashes appeared on the day following the injection, and their duration was from one to three days. They consisted in each case of fine, thickly-sown papules, which either gave rise to a coarsely punctate appearance, or fused together into well-defined patches, mixed with areas of simple erythema. In one case urticarial wheals were present. In some cases the whole body was more or less uniformly covered, in others the rash was chiefly apparent on the buttocks and extensor surfaces of the limbs, or on the trunk, especially at the sides.

It is not the object of this article to enter into an accurate description of enema rashes, or to discuss their diagnosis, but simply to point out the fact that no rashes whatever were apparent after the use of enemas made with soft soap. If the small number of cases mentioned in this paper does not justify the absolute assertion that soft soap enemas are not followed by rashes, it undoubtedly shows that these rashes are less frequent when soft soap instead of hard is employed for the manufacture of the injections.

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MILK AND ITS RELATION TO HEALTH AND DISEASE.*

By G. VIVIAN POORE, M.D., F.R.C.P.

I SHALL begin by recalling to the recollections of you all the great work that was done by Dr. Ballard, the medical officer of health for Islington, when in the year 1870 he demonstrated that an epidemic of enteric fever was definitely due to milk. Ballard's work was so important that I hope you will excuse me if I just recall the main actual facts with regard to it. They were these:—
During a moderate prevalence of enteric fever in Islington in the summer of 1870, Dr. Ballard noted that within a circle of less than a quarter of a mile radius there were 168 cases occurring in sixty-seven houses, and of these twenty-six died. As there were only twenty typhoid deaths registered in the whole of this large parish outside the special area, there was evidently some special cause for the existence of the fever within the special area. The house hygiene, which naturally first of all attracted attention, I think we may dismiss, by saying that the houses attacked by the disease were no worse than the others. Then the houses within the limited area where the disease occurred had the same water-supply as the rest of the parish and district. The theory as to the fever which found most credence amongst the inhabitants, and amongst some of the doctors too, was that it was in some way connected with the cutting made for the North London Railway. They talked of "miasmatic influences," and they said the soil was polluted. Dr. Ballard was told that the mother of one of the patients had an idea that the epidemic was due to milk, and Ballard frankly admitted that when the suggestion was made to him it appeared ridiculous. But he was a man of singularly open mind, and

* An address delivered before the Wimbledon Medical Society, November 8th, 1901.

he went to work with very great patience, and followed up the suggestion of the lady and made, with the willing assistance of the medical practitioners of the district, a house to house investigation. He found first of all that there were no less than eight cases of enteric fever amongst the family and employées of the dairy. Then he next found that fifty-four of the sixty-two families attacked (87 per cent.) were regular customers of the dairy, and of the twenty-six fatal cases one had come from the country and the remaining twenty-five were customers of the dairy. This particular dairy supplied 142 out of 2000 families living in the area, and it was found that seventy of these 142 families had been attacked, and that out of fourteen fatal cases occurring outside the special area five were regular customers of the dairyman in question, and others were chance customers, so that out of the total number of cases of enteric fever at that time in Islington, Ballard undoubtedly showed that a very undue proportion were customers of this dairy. He also found that in 75 per cent. of the families attacked the attacks were multiple; that is, that there were two or three, or even more, patients in one house. And in a particular house, where several families lived, and who all used the same foul privy, the only sufferers were an old man and an old woman who drank the suspected milk. Then Ballard went to the premises of the dairy, and an examination of the dairyman's premises showed that rats had established a communication between the sewers which received the fæces of the house first attacked with enteric fever, and a wooden subterranean water-tank, the water of which was used to swill the milk cans. The conclusion is inevitable.

I am perfectly well aware that everybody in this room is acquainted with this historic instance, but I have chosen nevertheless to give you the details of the case. It was the first time that milk as a serious cause of disease had been really brought into court, and I wish to recall to your recollection how patient was the work of Ballard, how he left not a stone unturned to prove his case. After Ballard's investigation of that Islington epidemic the conveyance of enteric by milk was no longer a "view" held by this man or by that man, but it was a fact, and you know very well that since Ballard pointed out this fact milk epidemics of

enteric fever have been frequently reported. It is due, I think, to the memory of the man who first really demonstrated this fact, that the whole circumstance should be accurately brought before you. It was indeed an epoch-making investigation by a man with a scientific and logical mind. He knew what care was necessary to prove his case, and he definitely proved it.

I would here point out that that epidemic, and others like it, were not due to anything wrong in the cow but were evidently due to bad sanitary arrangements. And I mention this because I am of opinion that there should be no difficulty whatever in any dairy farm, even in the wilds of the country, in obtaining an absolutely pure water supply. You cannot have a dairy farm unless some part of it be moderately near a stream. There are always meadows on the dairy farm, and to sink a well, properly made, with securely lined sides, and to dispose of the fæces by dry methods away from the well, is a perfectly easy thing, and there ought not to be any difficulty in getting such wholesome arrangements. It is my opinion and it is an opinion which may not be shared by all, that on a dairy farm subterranean sewage arrangements which are out of sight, and are liable to be invaded by rats and to leak into wells ought really, if we are to have any control, not to be allowed. The practice of swilling cowsheds and cow-houses with water and leaving them damp cannot be regarded as wholesome or sanitary in any way. You will find that the best and sweetest-smelling cowsheds are those without water laid on, where no swilling is possible, where the cleansing is carried out every day and the place swept, not swilled.

Now I wish to direct your attention to tubercular disease, and I wish to bring to your notice certain facts which have been brought forward as showing that there is a very definite connection between milk consumption and certain forms of tuberculosis. It is well known that in recent years death from phthisis and death from tuberculous disease have undergone a very notable decrease in this country. The only exception to this rule has been found in deaths from *tabes mesenterica* among children in the first year of life, and it was concluded by the late Sir Richard Thorne that as children presumably drink proportionately more milk in the first year than in any subsequent year,

it might be assumed that the cause of the increased mortality from *tabes mesenterica* in the first year of life was the bacillus contained in the milk coming from cows with tuberculous udders. This is a very large assumption. An assumption of that kind cannot be made and it cannot be proved without an immense deal of trouble, and I want to bring to your notice the facts as I have read them. In the ten years 1881—1890 there were 8,890,238 children born in England and Wales, and of these children the majority we may suppose were milk drinkers. I wish you to notice, please, that I am dealing with ten years and not with one. Dividing these milk-consuming babies into two classes, we find that whereas 39,194 died of *tabes mesenterica*, 8,861,144 did not die of *tabes mesenterica*, so that the odds against a new-born child dying of *tabes mesenterica* during the period of greatest milk-consuming appears to be 228 to one. Of the 8,890,238 children born, 1,259,860 died in their first year. That is 142 to every 1000, so that the odds are seven to one against a new-born child dying in its first year, but if it does die, the odds are thirty-two to one against its dying of *tabes*. If in a hundred milk-drinking children dying in their first year only three die of *tabes*, that does not give a high degree of probability that milk and *tabes* are cause and effect. Although the mortality of children in their first year slightly declined during the decade 1881—1890 (from 149 to 142 per 1000 births), it is still too high. It may be advisable to consider the chief causes of death of the 1,259,860 children who died in their first year in the ten years with which I am dealing. These were:—whooping cough and respiratory diseases 294,203; nervous diseases (chiefly convulsions), 215,665; diarrhoea, cholera, and digestive disease, 180,201; measles, 25,366; and no less than 21,095 died of "violence." *Tabes mesenterica*, as I have mentioned, killed 39,194, phthisis 7246, and other tuberculous diseases 32,615, the total of the tubercular group being 79,055, and other causes not classified killed 444,275. Thus it appears from these figures that tuberculous diseases caused at the outside about one in sixteen of the deaths of these milk-drinking babies as against about one in eight of the deaths of the entire population at all ages.

Now before it is possible to conclude that *tabes mesenterica* is a form of tuberculosis produced by milk-drinking it would be necessary to demon-

strate by post-mortem examination, that so-called *tabes* is in all cases tubercular. This does not seem in the least degree probable. We know very well that *tabes* is a loosely used pathological term, and that there are certain things which you cannot put on a death certificate unless you want to fight the parents. To take two things, you cannot put "starvation," and you cannot put "congenital syphilis," and I take it that *tabes* is often put down euphemistically because a child has wasted away, so that *tabes*, although a convenient term, is a term of low pathological value. It is impossible to conclude without a post-mortem examination that *tabes* is tubercular. Tubercular *tabes* is not diagnosable during life with anything like the certainty that enteric fever is diagnosable. Thus the question arises, out of the 39,194 deaths from *tabes mesenterica* how many were tubercular? The answer to that is not forthcoming. The next question which would have to be determined is, are the tuberculous mesenteric glands, supposing them to exist, due to a primary inoculation of the alimentary system? Most pathologists assert—German and English—that primary disease of the alimentary tract is not at all common, and that it is the rule rather than the exception to find caseous bronchial glands or other evidence of tubercle in the body, and if you find caseous bronchial glands or tuberculous lesions in the body, it is quite impossible to conclude that the condition of the mesenteric glands or the ulceration in the intestines is really primary.

Then supposing you satisfy yourself by post-mortem examination that the child had tuberculous disease of the glands, and that that tuberculous disease of the glands was primary, the next question you would have to decide is, to what extent was the child artificially fed? How much cow's milk did it get per diem? And did the milk contain tubercle bacilli? These questions would all have to be answered before you could give probability to the view that *tabes*, tuberculosis, and milk are related. Ballard found, as others have found, that the existence of his enteric fever was not amongst the very poor, but amongst the people who had money enough to buy lots of milk, and I do not think it would be the experience of most of us here present that the existence of *tabes* or the wasting of children was amongst the well-to-do. We shall

find it among the very poor indeed. Having found the bacilli in the milk, you would now have to determine whether or not the bacilli were alive, and did the bacilli come from a cow with a tuberculous udder, or from other sources? that is to say, had the cow a tuberculous udder, or did a phthisical milkman accidentally spit into the can? Those are all points which you have to decide, so that the question is a very, very difficult one to answer, and it cannot be done by merely holding "views." The probability is, as I think I have shown you by the figures quoted, that milk-drinking as a cause of tubercular diseases of the bowels is of no very great importance. Without a complete chain of circumstances, having a tuberculous udder at one end and tuberculous mesenteric glands at the other, it is impossible to establish even a low degree of probability that they are related as cause and effect. In how many of the 39,194 children who died of *tabes mesenterica* was such a chain established? And without such a chain being established I think the "view" is really nothing but a "view," and very hazy view too. Even with such a chain established is it possible to exclude other sources of infection in a child born into a world of which one-seventh part of the inhabitants are tubercular? So that you see one is surrounded with sources of fallacy on every side. Then having traced back the tubercle in the mesenteric gland of the dead child to the udder in the cow, can you start from the tubercular udder and say that round that tuberculous udder, or round the dairy, there were scattered in undue numbers cases of *tabes mesenterica*? I think that is a piece of evidence you cannot get, but it ought to be got. We are dealing with a chronic disease, a disease concerning the incubative period, of which we know nothing, a very insidious disease, and a disease totally different from enteric fever. Ballard showed that the cases of enteric fever could be traced back to the dairy, and he then went to the dairy and showed how the milk was infected, and how it spread the disease. He explained the matter from both ends. It is perhaps not possible to bring forward similar evidence in the case of *tabes mesenterica*. We ought, however, unless we can bring forward evidence of this kind, to maintain a discreet reticence, because it is no good "quarrelling with our bread and butter," nor with our milk

unless we have some real solid grounds to go upon. If, as seems probable, it be true that cow's milk as a cause of tuberculosis holds an insignificant position, I cannot think it is wise to adopt an alarmist's attitude on the question.

Now we will go to the death-rate from *tabes mesenterica*, and I find that the rate in 1881—1890 for the whole population—I cannot get it for children under one year of age—was '28 per thousand. Now certain counties were above the average for England and Wales as a whole, and these counties were Durham, Northumberland, Nottingham, London, Lancashire, the North Riding of Yorkshire, Staffordshire, and Worcestershire—all of them, you will perceive, manufacturing counties. When we come to the dairying counties we find that the rates for *tabes* are lower, and lowest of all is Dorsetshire where the rate was only '11 per thousand as against '46 in Durham, where the rate was highest, that is to say, there were more than four times as many deaths from *tabes* in Durham as in Dorsetshire. If you go through the agricultural and dairying counties you will find the same low figures, thus, Dorsetshire '11 per thousand, Rutland '12, Westmorland '13, Berkshire, Oxfordshire, Herefordshire, and Huntingdonshire '14, Somersetshire '15, Hertfordshire '15, Shropshire '15, Sussex and North Wales '18, Northampton '18, Devonshire, Gloucestershire, and Bedfordshire '19. It is an interesting fact that in the dairy counties the death-rate from *tabes mesenterica* is low and in the towns it is high. In short, proximity to the cow and possible facilities for obtaining raw milk does not seem to have had any untoward effect upon the *tabes* death-rate.

Next I will endeavour to show what is the incidence of phthisis in the milk trades. Dr. Tatham gives, in the supplement to the 55th Report, the death-rate of occupied males, or, rather, the mortality figures of occupied males between the ages of twenty-five and sixty-five from certain causes. I have only taken out some causes; I do not want to weary you with unnecessary details. The mortality figure from all causes in all males he takes as a thousand, and the mortality figure of occupied males is rather less, because all males includes diseased males as well as healthy ones, and the ones who are occupied live longer, of course. Now, the mortality figure of all occupied males from all causes, taking the mortality of all males

as 1000, is calculated by Dr. Tatham as 953; from alcoholism it is 13; from phthisis, 185; from bronchitis, 88; from Bright's disease, 27. The group of persons who have the highest mortality of any are the inn and hotel servants in London. Their mortality figure from all causes is 1971, that is, more than double the mortality figure of all occupied males; from alcoholism, 139, as against 13; from phthisis, 607, as against 185; from bronchitis, 153, as against 88; from Bright's disease, 60, as against 27. These figures bring out very well what Prof. Brouardel insisted on at the late Congress on Tuberculosis in London, that alcoholism is undoubtedly a great cause of death from tuberculosis, and the high mortality figure of 607 amongst hotel servants from consumption is a very interesting point. Now we come to the group that is most important to us, that is to say, the milk-sellers. The mortality figure of milk-sellers from all causes is 1061, which is a little above that of all occupied males. I may say that the mortality figure of all shopkeepers is above that of all occupied males. The figure for alcoholism is 16, for phthisis 166, that is, below the average for all occupied males. I think that is very important, that these people who are always about with milk have a mortality figure from phthisis less than that of all occupied males. All occupied males give 185, but the milk-sellers 166. Next I take the butcher. His mortality figure is 1096. From alcoholism, 35; phthisis, 195; bronchitis, 79; and Bright's disease, 35. Lastly comes the farm labourer. His mortality figure is very low. It is 632, and the deaths from phthisis only 115, and the farm-labourer is constantly engaged, of course, in milking cows, possibly with tuberculous udders, and probably manuring the land with the dung that is voided from the tuberculous intestines of cows. Taking the mortality figures for various trades from phthisis, we have printers, 326; book-binders, 325; hatters, 301; tobacconists, 280; tailors, 271; drapers, 260; shoemakers, 256; butchers, 195; milk-sellers and dairymen, 166; fishmongers, 160; greengrocers, 154; grocers, 131; gardeners, 106; physicians (who are always, if I may say so, in touch with tuberculous sputum, but they are driving in the open air and are well fed), 105; clergymen, 67. It is to be noted that in the above list those who are habitually brought presumably into close contact with the bacillus tuberculosis,

such as the physician who tends the patient, the clergyman who visits him and buries him, the dairyman who sells tuberculous milk and butter, the butcher who deals in the meat of tuberculous oxen, and the agriculturist who tends the tuberculous cows and spreads their tuberculous dung upon the soil, show no excessive tendency to phthisis. I will remind you these are figures culled from the tables of the Registrar-General in his supplement, and I think they are not to be lost sight of in examining this question.

Then, again, what has been our practice in this matter? Do we regard *tabes* in a child as due to milk or due to want of milk? It is a very great question indeed. At University College, where we have not too much money, we have a large Samaritan Fund, and we used to spend a good deal of that Samaritan Fund in giving away new milk to children that wanted it. We gave the child with *tabes* cod-liver oil, of course, but an order for a pint of new milk every day had remarkable effects, and the way those children thrived on it and the way they got well was truly wonderful. My experience at University College, and also at the Royal Infirmary for Women and Children, in the Waterloo Road, was exactly the same. I ask, then, whether we were giving meat or were we giving poison? If the latter certainly my impression was that the children thrived on it to a wonderful degree. It is a most important question. Now, I do not think that this question can be settled by laboratory experiments only, and I may say that, whatever the answer may be to the question which is to be tried again in the laboratories as to the exact relationship between bovine tuberculosis and human tuberculosis, we must not set aside the practical, the clinical, and the epidemiological aspects of the question.

I have asked again and again for epidemiological or clinical facts, which go to show that milk from a particular dairy has caused an undue amount of tuberculosis in its milk consumers. Such evidence is wanting. Perhaps that is due to the circumstances of the case and the nature of the disease. Be that as it may, until such evidence is forthcoming, we ought to be very careful how we move. We must have both science and practice going hand in hand. In Ballard's case the discovery of the bacillus entericus merely strengthened the attitude he had taken up. So with regard to

Lister's great work ; Lister took up what Pasteur had begun, and showed that Pasteur's views were facts, and facts capable of practical application. Now can you piece together milk and tabes mesenterica, and show that they are cause and effect?

It is too much the fashion at the present day to take up a bacillus and multiply it (as these things do multiply on cultivating media), and imagine all kinds of mischief. That is not science. That has been done a good deal of late years. For instance, people have said all bacilli are so dangerous that the only rational thing to do is to burn the dead body under all circumstances. Well, that is a matter that has interested me, and I have looked about for evidence of definite mischief having arisen from cemeteries. There is not any. I do not say that some illness may not have been caused occasionally, but on searching the Local Government Board Reports I get no such evidence. My own belief is that although cemeteries may be harmful, yet they do a great deal more good than harm, because they necessitate open spaces and growing of trees, which freshen the air, and they keep the population a little apart from each other. Our great object nowadays is to prevent undue density of population, and anything which prevents overcrowding and spreads the people ought to be very welcome. Then again people have said, and not very long ago either, that the typhoid bacillus grew in the earth. Well, it seems very natural that it should, but it does not, and there has never been a case in which a definite typhoid epidemic has been spread by the earth. They said this happened at Maidstone, but of course it did not. You have only to read the evidence carefully to find that such a statement is a perfectly gratuitous assertion. Six bacteriologists were at work at Maidstone, and they never discovered the typhoid bacillus in the soil: they had too much sense to try.

If I have not touched upon the burning question of the relation of bovine tuberculosis to human tuberculosis, it is because I am not qualified to speak on the subject; I am not a bacteriologist, and it is purely a bacteriological question. But even supposing that by dint of great ingenuity, and energy, and perseverance, you can make a bovine tubercle bacillus grow in a human subject, we should still want to know the meaning of it, and

we must remember that just now people are very busy taking a disease from calves and inoculating it into human beings *to do them good*. Lord Lister said at the Congress on Tuberculosis that it had been very difficult to prove that vaccinia and variola are the same disease. Even if the possibility of the transmission of bovine tuberculosis to the human subject be admitted, you have still got the other question to answer, whether such transmission works mischief to the human race. I feel that we must keep to the practical and clinical side, and ask ourselves whether we in our daily experience have definitely seen harm from drinking good milk—I am not speaking of sour milk or bad milk. I think it is an important matter, because we must be careful in this country not to get under the influence of doctrinaires, of people who start a particular view, and then run it for all it is worth and write it up. I have brought figures forward which go to prove that the whole thing needs a very careful consideration, and that evidence is wanting from many sides.

The chemist can tell us very little indeed about food. Whether it contains so much nitrogen or so much carbon, etc. is not of much interest. There is a great deal in food besides its chemical composition, and I would remind you that we know nothing about the chemical cause of scurvy. All chemical theories we are inclined to set aside altogether, and we go back and say it is the want of fresh food—fresh vegetables and fresh meat. If we use these with really fresh milk from the cow, or from the breast, scurvy tends to disappear. When we are told that sterilised milk is as good as new milk for the child because animals maintain their weight on it, I think that is a specimen of science run mad. Children fed upon artificial food maintain their weight, but though they may be fat, rosy, and plump, they may soon be down with rickets or with scurvy, so that the mere fact that the child maintains its weight is not enough. How about its vulnerability to disease of various kinds? Remember that to us or to children over nine months of age who are beginning to take a mixed diet, the question of whether the milk is boiled or fresh is not possibly of much importance. It does not matter to me whether my milk is boiled or not; I do not consume many pints in a month, and I am getting a mixed diet of many other

things. But to the sucklings, whether they get the right or the wrong thing is very important, and we have to remember that we cannot find that out in a few months or weeks. It may take several generations, but nevertheless it is extremely important, and all the more so, because with the increase in population of our towns and cities, the inhabitants are getting further and further removed from their milk supply and from their food supply. We know now practically nothing about our food supplies, and we get to know less and less about them except that most of them are wonderfully and curiously made. You must remember that sterilised milk is all in favour of the dairyman; sterilised milk will keep for a month. He bottles it, but is the bottled fluid the same as fresh milk? I doubt it very much indeed. The same remark applies to meat, which nowadays is often killed at the Antipodes. Is such meat the same as fresh-killed meat? We get more and more preserved foods, and we get further and further away from the really fresh foods. There is a great deal more tuberculosis in the cities than in the country. I am inclined to think that the wasting children of the cities will be improved by giving them as much new milk as you can get them to take.

Neuritis caused by Occupation.—L. Hoeffmayer.—A professional neurosis usually develops on a neuropathic basis, but the neuritis in the cases described occurred in robust men, compelled to use the right arm in planing or polishing, etc. It affected the region of the brachial plexus and simulated rheumatism in the shoulder and arm, most severe at night, and induced by certain movements of the arm. Anti-rheumatic remedies had no effect, and there were no signs of myositis. The pains and tenderness on pressure were localised at the points where the nerves involved were accessible. All recovered after treatment by rest, heat, and galvanisation. Application of leeches to the most sensitive points also proved useful in some cases. Quinine and phenacetin relieved the pains at night, and rendered morphine unnecessary. The neuritis was cured in the course of eight or nine weeks, but the patients were advised to seek some other occupation for the following six to twelve months.—*Journ. Amer. Med. Assoc.*, December 17th, 1901.

CLINICAL LECTURE

ON

“SOME CASES OF OVARIOTOMY.”

Delivered July 5th, 1901, at the Royal Free Hospital.

By JAMES BERRY, B.S., F.R.C.S.

(Concluded from p. 173.)

OVARIAN tumours that come under the care of the general surgeon are usually large ones, forming visible abdominal swellings. The smaller ones, occupying the pelvis only, more often come under the care of the gynaecologist; at least, such is my own experience.

The diagnosis of large ovarian tumours generally has to be made (1) from uterine tumours (including pregnancy) and distension of the bladder, in both of which the tumour, like the ovarian tumour, has a pelvic attachment and grows upwards; (2) from tumours having no obvious pelvic attachments, such as large cysts of the kidney (hydronephrosis) or of the pancreas, and certain tumours, both cystic and solid, of the omentum or mesentery. The fact that these do not tend to occupy the pelvis or displace the pelvic viscera will generally, however, afford a clue to the diagnosis. It should not be forgotten, however, that an ovarian tumour may occupy an unusually high position and thus come to simulate the above-mentioned tumours. I remember on one occasion, some years ago, making a *post-mortem* examination on a case in which an ovarian cyst had been explored from the loin under the impression that it was a hydronephrosis. The septic sinus that resulted from this was the chief cause of the death that occurred when subsequently an ovariectomy was performed.

The diagnosis between large ovarian tumours and collections of fluid in the peritoneal cavity has already been mentioned. I ought, perhaps, also to mention that a very large hydatid in the lower part of the abdomen or pelvis might easily be mistaken for a true ovarian cyst; we have lately seen an instance of this in one of the medical wards of this hospital.

I have already laid stress upon the importance of not mistaking a gravid uterus for an ovarian cyst, a mistake which is by no means very uncommon. Only a few days ago I heard of a case in which the appearance of the legs of the foetus

was the first intimation received by the surgeon that he was not dealing with an ovarian cyst. To mistake a normal gravid uterus of large size for an ovarian cyst can only be the result of gross carelessness and insufficient examination on the part of the surgeon. Sometimes an erroneous menstrual history, and the omission of an examination of the breasts may lead to such a mistake. More often, however, it is to be hoped, the error arises from some abnormal condition of the pregnant uterus itself. It may occupy an unduly high position, or be unduly distended, as by hydramnios. It is well to remember, however, that even if the abdomen has unfortunately been opened by error in such a case, the true nature of the tumour ought to be recognised even then, before any serious harm is inflicted upon the patient, by observing its colour. The dull red colour of the uterus is very different from that of any ordinary ovarian tumour.

The difficulty of diagnosis may, however, be increased when, as frequently happens, pregnancy co-exists with ovarian tumour. The following case, which occurred in my wards a few months ago, illustrates this point:—A woman, æt. 25, who had been married two months, and who gave a history that her menstruation had ceased five months ago, was sent up to me from Lincolnshire with a greatly distended and very tender abdomen. The swelling had existed for five months, but had become very much larger in the last three weeks, during which time she had suffered acute pain. The abdomen was so much distended and so tense that the presence of a very large fluid-containing tumour, certainly ovarian, was at first all that could be diagnosed. No second tumour could be made out through the abdominal wall until the patient was under an anæsthetic; it was then easy to see and feel that there were at least two tumours, one a large ovarian at the upper part of the abdomen, the other, a firmer and more resisting mass, extending up to the umbilicus, and looking and feeling like a uterus of six or seven months gravidity. The breasts, however, although highly suggestive of pregnancy, were still rather small, and not quite like those of so advanced a pregnancy. The os uteri was soft and pushed forwards by a tense, round, immoveable mass in Douglas's pouch, which was apparently neither a retroverted uterus (the symptoms of which were moreover absent) nor an

extra-uterine pregnancy. It seemed clear, therefore, that there was pregnancy together with one large ovarian tumour and a second smaller tumour of uncertain nature in the pelvis. I made the abdominal incision a good deal higher than usual in order to avoid the uterus, and then found (1) a large ovarian cystoma much inflamed; this was tapped and removed in the usual way; (2) a gravid uterus of about five months, which had been pushed up into an unusually high position by (3) a smaller ovarian cyst impacted in and occupying the whole pelvis. The removal of the second cyst was a little difficult as I was anxious if possible to avoid disturbing the uterus much, lest abortion should follow. Abortion, however, fortunately did not take place, and the patient returned home quite well three weeks later.*

One more case illustrating the diagnosis between uterine and ovarian tumours, and I leave this part of my subject.

Lobulated fibroid weighing eighteen pounds; hysterectomy; recovery.—In October last I was asked by Dr. Wilson of Hackney to see with him a lady, æt. 52, who had a large abdominal tumour which gave her considerable trouble. The history was that for eight or nine years she had had a large hard tumour on the right side of her abdomen, which caused her no trouble. Three months ago she first noticed another tumour on the left side, which had grown rapidly to a large size. During this time she had had much trouble in micturating, and the bulk and weight of the tumours had caused much breathlessness, and prevented her from getting about or doing her work. Menstruation had always been regular but excessive until five months ago, since which time it had nearly ceased. The abdomen was greatly distended by two tumours, which nearly filled it. That on the left side was as large as a man's head and rather soft; the other was smaller and much harder. The tumours could be moved upon each other to a considerable extent. Vaginal examination showed a rounded, firm mass high up in Douglas's pouch. The os uteri was out of reach, and a sound was therefore not passed.

The diagnosis of uterine fibroid was easily made as regards the smaller abdominal tumour: the nature of the larger soft one, in view of the history

* The pregnancy progressed normally, and the patient was delivered at home of a full-term child four months after she had been admitted to the hospital.

of apparent rapid growth, was not so easy; the diagnosis lay between a soft fibroid or fibrocystic tumour, an ovarian tumour, and, possibly, some malignant growth. As the tumours were causing great trouble, exploratory abdominal section and, if possible, removal of the tumours were recommended and readily agreed to by the patient. At the operation I found that the tumours were all fibroids, some soft, some hard, completely burying the uterus within them. Hysterectomy was performed by Doyen's method without any difficulty, and the patient made a good recovery, leaving the nursing home on the nineteenth day. She has remained perfectly well ever since. The tumours after removal weighed eighteen pounds.

To mistake a distended bladder for an ovarian cyst seems at first sight such an unlikely error that I should scarcely have thought it worth while to mention it had I not actually seen this mistake made on one occasion. A few years ago I had to make a *post-mortem* examination at another hospital upon a woman who had been admitted for a pelvic uterine fibroid and a large hypogastric tumour. The latter had been diagnosed as an ovarian cyst, and it was not until the abdomen had been opened that the true nature of the case was revealed. The patient, however, gradually sank and died about three weeks later. In this case the fibroid had pushed the bladder upwards and probably caused some obstruction to the outflow of urine.

The moral of this case is that you cannot be too careful to see that the bladder has been thoroughly emptied before you undertake an ovariectomy.

I should like here to offer you a word of warning as to the diagnosis between small inflamed ovarian cysts and appendicitis. In these days, when we hear so much of appendicitis, we are a little apt to jump to the conclusion that a young woman who has had many attacks of pain in the right iliac fossa, with, perhaps, tenderness and increased resistance in that region, is suffering from appendicitis. We may be unwilling or unable to make a thorough pelvic examination, and mistakes may thus arise. Even when a thorough pelvic examination has been made, it may be very difficult to distinguish between the mass of inflamed tissues surrounding a diseased appendix (especially if this happens to lie within the true pelvis) and the inflammation surrounding a suppurating or inflamed ovary or tube. Moreover, an appendix not itself diseased

is very likely to become adherent to any inflamed structure in its neighbourhood, and thus to increase the difficulty in diagnosis.

It is well, therefore, before making the diagnosis of chronic appendicitis in a woman, to consider carefully whether the symptoms may not be due to disease of some pelvic organ rather than to the appendix itself.

I have myself seen more than one case in which a supposed appendicitis proved at the operation to be a case of small inflamed ovarian cyst. I have also seen at least one case in which a well-known abdominal surgeon made the converse mistake, undertaking what he believed would be an ovariectomy, and finding when the abdomen was opened that he had to deal, not with an ovarian tumour, but with a chronic abscess connected with appendicitis.

In connection with this subject, I will relate briefly a case which some of you saw in my wards about a year ago.

An unmarried woman, æt. 28, was sent up to me from the country with the history that for eight years she had been subject to severe attacks of pain in the right iliac fossa. These attacks had of late been getting more and more severe, and she had often been confined to her bed for several weeks at a time. Tenderness and a deep-seated swelling near the brim of the pelvis had been noticed during the attacks. The diagnosis of appendicitis had been made. On making a pelvic examination I found that a considerable fixed, tender mass was easily felt high up on the right side of the pelvis, and extending into the iliac fossa. I was uncertain whether this mass was an abscess of the appendix extending down into the pelvis, or whether it was primarily a disease of a pelvic organ, and I therefore refrained from making any definite diagnosis. At the operation I found a small ovarian cyst quite full of pus, and universally and firmly adherent to all the surrounding structures. The appendix itself was firmly fixed to the cyst, and contained two fæcal concretions, but was otherwise quite healthy. The cyst was dissected out with some little difficulty, owing to the extent and density of the adhesions, and the patient then made a good recovery.

Another class of cyst, distinct from dermoid and from true ovarian cysts, is the parovarian, of

which I will cite to you but a single instance from my own practice.

[A typical case was here described, and reference made to the difference between the thin watery fluid contained in these cysts and the viscid fluid of an ordinary ovarian tumour.]

Solid innocent tumours of the ovary are not common. A case of this kind came under my care a year or two ago. A middle-aged woman presented herself with symptoms of chronic intestinal obstruction. On examination by the rectum an intensely hard smooth mass was found occupying Douglas's pouch. It was so firmly fixed that some doubt was felt whether it might not be a tumour springing from the wall of the pelvis. The passage of a uterine sound showed the uterus to move apart from the tumour; there was no menstrual history suggesting fibroid. Owing to my indisposition at the time, the actual operation was kindly performed for me by a colleague. The tumour which you see here proved to be a dense fibroma of the ovary about as large as a goose's egg.

I remember seeing a case under the care of another surgeon a few years ago in which both ovaries were the seat of a similar fibrous tumour about as large as an egg.

Malignant ovarian tumours are not uncommon, but it so happens that but very few have come under my own care.

The following is an example of a large solid tumour, which the microscope showed to be malignant, although the course pursued by it was not very rapid, showing that its degree of malignancy was low:

In May, 1899, I went down into the country to operate on a lady, *æt.* 58, who had been feeling ill for about a year, and had had vague abdominal symptoms of discomfort. The occurrence of some irregular uterine hæmorrhage led to an examination by her doctor, who discovered a large smooth abdominal tumour, nearly filling the pelvis, and extending upwards as high as the umbilicus. It was not connected with the uterus. On opening the abdomen a dense, solid, slightly nodular tumour presented itself. At first sight it appeared to be fixed, and to be hopelessly irremovable. Further examination, however, showed that its apparent fixity was merely due to its being jammed in the pelvis, and after some little trouble I succeeded in

dislocating and removing it entire. It was attached by a comparatively narrow pedicle. It had nowhere actually penetrated its capsule, and no secondary growths were discovered. The tumour, which you see in this jar, is about as large as the head of a newly-born child, and is solid throughout. When I last saw this patient, some months ago, she was still in excellent health, and there were no signs of recurrence.

I should like to tell you of other cases that I have had, some of which are also not without interest, but I fear this lecture has already been unduly long, so I pass them by. The time at my disposal does not allow me to discuss at any length the subject of treatment. I would merely say that judging from my own operations, and from the far larger number of those that I have seen performed by other surgeons, the chief points to which attention should be paid are the following:

(1) The most rigid asepsis throughout the operation.

(2) The avoidance of irrigation of the abdominal cavity, except in very rare and exceptional cases.

(3) The avoidance of the introduction of anti-septic solutions into the abdominal cavity; local sponging and the use of sterile saline solution are preferable to treatment by more irritating lotions in those cases in which contamination from pus has occurred.

(4) Drainage is seldom desirable, but if it is necessary, strips of gauze are better for this purpose than any other material.

An ordinary ovariectomy is a very simple affair and should, if properly performed, be attended with a very low mortality indeed. It is in the treatment of the cases complicated by peritonitis, suppuration, extensive adhesions, malignancy, and so forth, that difficulties and dangers become prominent. With the view of ascertaining what results can be obtained by skilled operators who have had a far larger experience of the operation than I have myself, I have obtained, by the kindness of the house surgeon at the New Hospital for Women, the records of the ovariectomies performed there in the last few years. During the years 1895—1900, eighty-three ovariectomies were performed; all the patients recovered except one who had a large ovarian cyst, and who was found, at the time of operation, to have also malignant disease of the stomach, of which she died about a week

later. For the details of all these cases, however, I must refer you to Mrs. Scharlieb and her colleagues, with whose practice some of you are already familiar. With regard to my own results, the total number of my operations has been comparatively small, but I am glad to be able to state that, with one exception, I have never yet lost a patient after an ovariotomy, nor have I ever had any serious after-complication of any kind. With regard to the one exception to which I have alluded, and which I propose now to relate to you, I think you will agree with me that I may fairly claim that the ovariotomy had but little to do with the death. The patient was a married woman, *æt.* 25, admitted on account of carcinoma of the rectum; she was in an early stage of pregnancy, and had also an ovarian tumour as large as an ordinary cocoanut, which was impacted in the pelvis. The carcinoma admitted of removal, so a preliminary colotomy was performed. At the same time, through another incision, the impacted ovarian tumour was removed, as it was feared that if left it would seriously interfere with the pregnancy and subsequent delivery. The patient had no bad symptoms of any kind after this operation, and three weeks later excision of the rectum was performed. This operation, which was a severe one, was unfortunately followed a few days later by abortion and fatal peritonitis.

In order to avoid, as far as possible, bad results after ovariotomy, it is well to know what causes are likely to produce death after this operation. It will, perhaps, be interesting and instructive to you if I review briefly all the fatal cases of ovariotomy that came under my notice during the five years in which I held the post of surgical registrar at St. Bartholomew's Hospital. It was at that time part of my duty to make *post-mortem* examinations, not only on all patients who had died in the general surgical wards, but also on those from the surgical side of the gynaecological wards. The experience thus gained was to me both interesting and instructive. During the above-mentioned period 176 ovariotomies were performed. Twenty-two patients died, and in nearly every case a *post-mortem* examination was obtained.

Two patients died of intestinal obstruction; in three cases the tumour was a suppurating cyst; in two cases the cyst had ruptured before operation;

one was a case of inflamed cyst; one was a large universally adherent cyst; one was an adherent dermoid tumour; in two cases the pedicle of the tumour had been twisted; in two cases the tumour was malignant; in one case an opening into the rectum was found; in another a sinus had existed in the loin (this case has already been mentioned); one patient died suddenly of collapse and high temperature on the second day; one died of bronchitis five weeks after the operation; two died of peritonitis, for which no cause could be discovered at the post-mortem; and two died of calculous pyonephrosis and carcinoma of the stomach respectively, complications which, although existing at the time of operation, had not been detected. Further details of all these cases will be found in the published hospital reports of the period mentioned.

Heredity in Appendicitis.—F. Forschheimer, in 'American Medicine' of October 5th, 1901, calls attention to the fact that the heredity of appendicitis receives no mention in American medical literature. Foreign writers have reported a number of families in which this disease was frequent. Talamon says that the reason this subject has been overlooked in medicine is due to the fact that the clinical forms and methods of treatment of appendicitis have been discussed, to the neglect of the *ætiology*.

Forschheimer reports three family histories in which appendicitis and bowel disturbance were very common. In the first family there was a train of symptoms going through all the members of the second generation. These refer to gastrointestinal disorders associated with nervous symptoms and circulatory disturbance. In the third generation there was appendicitis. In the second family the father probably had appendicitis, and he as well as the mother had constipation. All the members of the second generation had some gastro-intestinal disturbance. Three of the members of this family had appendicitis. In the third generation of the same family constipation was the rule, and in the fourth generation there were two cases of gastrectasis. The explanation of these conditions the writer finds in some inherent weakness of the bowel as a whole, deficiency in its muscular development and nerve function, or possibly an inherited form of vicious metabolism, plus the inherited condition of the appendix.—*Medicine*, December, 1901.

A CLINICAL LECTURE
ON THE
DIAGNOSIS OF TUBERCULOSIS.

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GENTLEMEN,—Difficulty of diagnosis in cases of tuberculosis is often very great, and this whatever organ may be the seat of lesion.

In medical practice tuberculosis of the chest, the abdomen, and the head is most frequently met with; in not a few cases all of these cavities may be affected, but as a rule the symptoms for which relief is sought are more or less prominent in one or other of the great divisions just alluded to. It may be said, in fact, that general tuberculosis tends to chiefly attack one organ or system of organs, while at the same time a similar lesion is, to a greater or lesser extent, universally diffused throughout the body, but this diffusion does not declare itself by any symptom or physical signs.

On the other hand, it may be that marked physical signs appear in the course of time in the case of organs which, at the first onset of the illness, were entirely free from any manifestation of disease, and when this is the case much light may be thrown upon the diagnosis which may hitherto have been obscure or equivocal.

I recall the case of a boy *æt.* 18 whose health had always been excellent and whose family history was most satisfactory. He had lived all his life in a healthy country neighbourhood, and had constantly been out of doors. Without any apparent cause he began, six months before admission, to pass blood in the urine, not continuously, but from time to time. There was no other symptom. The urine apparently contained no pus, and was in nowise abnormal except as regards the presence of blood. At this time his lungs were perfectly healthy, and it was thought that the case might be one of stone in the bladder.

He was examined surgically, the bladder being sounded, but no stone was found. When I saw him he was very thin, but made no complaint beyond that of the passage of blood in the urine. Examination of the chest revealed harsh breathing, and some loss of percussion resonance at

both apices. A few weeks later the temperature began to rise at night, and at the same time the chest signs became far more marked; indeed, in another three weeks definite dulness with moist sounds was obtainable at both apices and symptoms of hectic fever became prominent. The urine now contained not only blood, but also pus, and on examination of the latter numerous tubercle bacilli were detected.

It was perfectly clear that the case was one of acute tuberculosis, and the result proved the correctness of this view of the nature of the illness. Yet for many months this young man's malady pointed for a time more strongly to the diagnosis of a local bladder affection than to the disease which was actually present, viz. a tubercular lesion of the kidney.

The entire absence of pus in the urine at the early stage of the malady was a strong point against the diagnosis of tubercular renal disease, and until the examination of the urine for tubercle bacilli, it was not possible to make an accurate diagnosis, apart from the fact that by this time the pulmonary physical signs and symptoms had thrown a flood of light on the nature of the disease.

A history such as this is very instructive, and should make us hesitate in giving too early a positive opinion in cases of hæmaturia; certainly it might have been thought that the healthy surroundings and family history made the diagnosis of tubercular disease most unlikely. In other words, almost any explanation of the symptoms seemed more probable than the genuine one, for the case had far more the appearance of stone in the kidney or in the bladder, or of growth in the latter, than of the hopeless disease really present.

Some years ago a girl *æt.* 10 was brought to me in the out-patient room with the following history:—She had been in her usual good health until ten days before being seen. At the time mentioned she began to complain of sore throat, together with slight difficulty in swallowing. During the ten days of throat affection her mother had noticed that the child was languid, quiet, and inclined to sleep during the day, but she did not take to bed.

When examined in the out-patient room it was found that there were patches in the fauces; the

tonsils were enlarged, the uvula was adherent to the right tonsil, and both tonsils and uvula were covered with patches of what appeared to be yellow tenacious membrane, which, on being forcibly detached, left a red and bleeding surface.

The temperature was no more than 99.6° , and the only complaint was that of debility, together with hoarseness of voice and difficulty of swallowing. It should be particularly noted that there was no albuminuria. The glands at the angle of the jaw on both sides were distinctly swollen and tender.

Now, here was a group of symptoms and physical signs which strongly pointed to the diagnosis of diphtheria. This diagnosis was totally incorrect. Some days later the child died, and at the post-mortem examination it was found that the lungs were stuffed with miliary tubercle from base to apex, and at the left upper lobe were several small, newly formed cavities. The fauces were covered with scattered tubercle of greyish-yellow colour, and the whole interior of the larynx was in a similar condition, but here the size of the tubercles did not exceed that of a pin's head, and they were arranged in a continuous sheet.

In each vocal cord, and symmetrically placed, was a small ulcer.

There were a few tubercles beneath the capsule of the spleen, none on the liver or pericardium.

In nearly the whole length of the ileum there were numerous characteristic tubercular ulcers, and the ileo-cæcal valve was the seat of similar extensive ulcerations on its upper aspect. The large intestine was healthy throughout.

It should be mentioned that in this case frequent attacks occurred in which much embarrassment of breathing was noticed. So great was the obstruction to the entrance of air to the chest that both inspiratory stridor and marked drawing in of the lower chest-wall was evident during the attacks.

It seemed that tracheotomy might at any moment be necessary.

The case was one of acute tuberculosis, in which the extreme virulence of the disease manifested itself in the throat and larynx. Yet, clinically, it had all the aspects of diphtheria, with marked involvement of the larynx.

On criticising the diagnosis of diphtheria it is necessary to remark that the absence of albumin from the urine was a point to which perhaps not

sufficient weight was given. My experience has been that in practically every case of genuine diphtheria albuminuria is present at some period of the illness, and in nearly every case it has been perfectly obvious when the patient was first seen.

Further, although laryngeal symptoms may occur at any age, they are far more frequent in quite young children and infants; in fact, laryngeal diphtheria is certainly rare after the age of three or four.

This child had passed that age, and more attention might have been given to this point.

It might be said that the child had been ill too long for the case to be one of diphtheria; but in taking histories, especially in hospital patients, it must never be forgotten that the powers of observation are ill-developed, and the intelligence most limited. Therefore it is not prudent to attach much importance to such a statement as that the child had been ill ten days, though even this lapse of time would not be incompatible with the condition and appearances actually observed. As regards the languor and indifference, they are characteristic of both maladies.

The temperature (99.6°) when the child was first seen, was quite consistent with the diagnosis of diphtheria.

It might be said that surely an examination of the chest would have thrown a flood of light upon the nature of the case. Nothing was noticed on stethoscopic examination beyond the presence of marked rhonchus and stridor. No crepitation was anywhere audible, and no dulness could be detected over any part of the chest-wall. This is, of course, what is generally found in cases of acute pulmonary tuberculosis; in this deceitful malady the chest physical signs are simply those of bronchitis.

In connection with this subject I recall a case in which the patient, a woman of middle age, came to the out-patient room with the complaint of troublesome cough of some months' duration, and which gradually got worse. Rhonchus and sibilus were audible over the whole chest, back and front, but there were no moist sounds; neither was there any dulness. In fact, the case presented the features of acute bronchial catarrh. But the lips of the patient were cyanosed, and the breathing was rapid, out of all proportion to the severity of the chest lesion.

Acute tuberculosis was diagnosed, and the diagnosis was found to be correct. On post-mortem examination some weeks later, the lungs were full of closely packed miliary tubercle.

Small children may appear to be suffering from an attack of acute bronchitis, but at the same time undue accentuation of the respiration and some œdema of the extremities may also be observed. When this is the case, the great probability of the case being one of acute tuberculosis should never be lost sight of; if this is overlooked disastrous mistakes in prognosis will inevitably result.

The great difficulty of differentiating typhoid fever and acute tuberculosis is well known.

A girl of eighteen came to me in the out-patient room, stating that for ten days previously she had been out of sorts, with pain in the limbs, headache, and weakness. When first seen she was in a hysterical condition, was very prostrate, and wandering mentally. She was at once admitted, and the case was regarded as one of enteric fever at the end of the second, or beginning of the third week.

The temperature was continuously high. Five days later she died, and the post-mortem examination proved the case to be one of acute tuberculosis.

The left lung was full of grey miliary tubercle, the nodules being of the average size of a hemp seed; some of the tubercles were in the softening stage. The pleura was abundantly studded with tubercle.

The right lung was in a similar condition, and there were several calcified caseous glands in the mediastinum. There was an abundant deposit of miliary tubercle between the diaphragm and the upper surface of the liver. The small intestine was the seat of tubercular ulceration.

In cases such as the one just described it is really impossible to be certain of the diagnosis; they are most often observed in quite young children, but may occur at any age. It is obvious that any laboratory test which is helpful in the diagnosis of these difficult cases would be very welcome, and there certainly are tests which are recommended for clinical use. Unfortunately, neither the occurrence of a positive result with the diazo-reaction, nor with that of Widal, is absolutely conclusive as to the nature of the case

under observation. My own experience of the diazo-reaction leads me to conclude that it is practically always present in cases of enteric fever, during, at all events, some part of the course of the disease, but it certainly also gives a positive result in tuberculosis. Thus it fails just at the point where its help would be of the greatest use.

Much the same remarks apply to the Widal reaction, which is undeniably open to the objection that it reacts positively in cases other than those of enteric fever.

The lesson should not be lost that, after all, it is better to trust to the unaided methods of examination and to the results of experience than to rely unduly upon any laboratory test whatsoever. In my opinion the tendency, so marked at the present time, to rely upon special tests and special modes of examination, not excluding the employment of the Röntgen rays in medical practice, is much to be deprecated. The student of to-day is taught to be far too reliant upon these methods, and the inevitable result will be the neglect of that experience and study of ordinary modes of investigation which, after all, are far more reliable than any special instrumental or chemical contrivance whatever.

From time to time cases are observed in which typhoid fever simulates the symptomatology of tuberculosis. The following history well illustrates this curious tendency of one disease to assume the clinical aspect of another and quite different malady.

A man was well and at work when he, quite suddenly, complained of severe headache, chilliness and weakness. When seen in the out-patient room he had been ill for some days, and had left his bed in order to come to the hospital. Constant sickness had been complained of, but no diarrhœa.

His temperature was 102°. There was constant sickness and the abdomen was much retracted, in fact, scooped out. His manner was very apathetic and he lay low in bed on his back. The very slightest touch elicited a marked *tache cérébrale*. Although the fever was somewhat high the pulse seldom exceeded 88; it was, moreover, very irregular. The sickness persisted for a week, as did also the irregularity of the pulse and the extreme retraction of the abdomen. At the end of this time the usual symptoms of enteric fever appeared—diar-

rhœa, rose spots, and distension of the abdomen. The patient died of perforative peritonitis, and post mortem, the usual lesions indicative of typhoid fever were found to be present.

This case was one of peculiar difficulty as regards diagnosis.

The sudden onset, the comparatively slow (as regards temperature) and irregular pulse, together with the extreme retraction of the abdomen give the case anything but the aspect of enteric fever. As regards the two latter features, indeed, they pointed most strongly to tubercular meningitis. And the sudden onset of the disease is most remarkable, for if there is one malady in which the symptoms commence insidiously that disease is typhoid fever. On the other hand, the suddenness of the onset in no respect militates against the diagnosis of tubercular meningitis, for in many cases of the latter malady some sudden head pain, spasm, or some mental change may be the very earliest indication of the disease that is impending.

Not seldom cases are met with in which abdominal symptoms have followed upon some accident involving a violent blow which has been struck on the abdomen. All kinds of views are held as to the causation of the symptoms in such cases, of which the presence of deep-seated abscess and chronic peritonitis are the most usual.

The diagnosis is entirely wrong, for in every case of this kind which has come under my own observation the accident has been a mere coincidence, having in reality no connection whatever with the disease, which has invariably been tubercular peritonitis.

I have seen cases in which the tubercular affection has been transmitted from the vesiculæ seminales, which were the primary focus of the malady; in others it has been the epididymis which has been the seat of the tubercular deposit.

When the vesiculæ seminales are involved in the tubercular process it is not unusual for the patient to complain of a periodical purulent discharge from the urethra; this, when present, is a most important factor in making the diagnosis, and one, too, which is especially liable to be misinterpreted.

In nearly every case of tubercular peritonitis which it has been my lot to observe there has been at some period of the disease a tendency to inflammatory redness and swelling of the skin round the umbilicus.

This is an important evidence of the presence of tuberculosis of the peritoneum, and is a physical sign which should always be carefully sought for, and, when found, allowed to have great weight in the determination of the diagnosis.

Tubercular peritonitis is generally of insidious onset, the patient when first coming under observation having been complaining of ill-health, abdominal pain, weakness, sweating, etc., probably for weeks or months previously. But that the malady may have a totally different mode of evolution is well shown by the following history:

A man of twenty-three came to me in the outpatient room, stating that until fourteen days previously he had been in his usual good health. At that time, and without apparent cause, he began to complain of pain in the abdomen, of gripping character, and chiefly around the umbilicus. There had been no diarrhœa, but he had been sick occasionally.

He had taken to his bed about five days before coming to hospital.

His previous health had always been very good, but he had been in the habit of drinking to excess. The family history was unimportant. His facial aspect was that of peritonitis. The abdomen was much distended and tense, being also markedly tender on pressure. The respiration was thoracic. A very obvious thrill was detectable in the abdomen, and dulness in both flanks, very far back, was obtainable.

The presence of fluid in both pleural cavities, and the rapid increase in the quantity of the same, threw much light on the diagnosis, which was that of tubercular peritonitis.

Death ensued in a few days, and, post mortem, the diagnosis was fully confirmed.

The intestines were so matted together in this instance that it was wholly impossible to unravel the tangled mass. The thickness of the parietal peritoneum was quite a quarter of an inch, and the great omentum was converted into a dense, cystic mass, very resistant to pressure, stretching across the upper portion of the abdominal cavity.

Now, when first seen, this patient presented the features of subacute peritonitis, and the history being so recent it seemed far more likely that the symptoms were due to perforation of some portion of the alimentary tract than to tuberculosis of the peritoneum. Had it not been for the fact of the

presence of double pleural effusion it seems very probable that the diagnosis might have been altogether wrong.

A singular point in the case is the contrast between the chronicity of the lesions and the acuteness of the symptomatology. It seems incredible that lesions, obviously of old standing, such as those of the peritoneum and great omentum, should give rise to no symptoms whatever during the long period which must have elapsed between their commencement and the onset of the pain for which he sought advice.

I have not much to say as regards the subject of tuberculosis affecting the brain and its membrane. The insidious onset of tubercular meningitis is, of course, perfectly familiar to all, and yet this fact is worthy of emphasis. The beginnings of this fatal malady are often apparently most trivial, and are extremely likely to pass unnoticed at the time. This is especially apt to be the case when the disease occurs in the persons of older people, those who have passed the age at which tubercular meningitis is most often seen.

In the case of a young man of twenty-three, for instance, extreme irritability, a most unusual occurrence with him, was the very earliest brain symptom of what was impending; within a fortnight he was dead. In another case a youth of nineteen had a favourite canary; he was fond of the bird, and pleased when it sang. Suddenly he took a violent aversion to the singing and threatened to wring the bird's neck. He, too, in about ten days was dead of tubercular meningitis, the symptoms being most typical, and the diagnosis confirmed by post-mortem examination. In the case of a young child the only symptom observed at the earliest period of the attack was persistent priapism.

It should be noted that in not a few of the cases of tubercular meningitis a persistent discharge from one or other ear is often present. This complication may lead to the diagnosis of brain affection consecutive to ear disease, and might even point to the desirability of surgical interference for the supposed aural trouble. A careful enquiry into the history of the case, and a painstaking observation of the symptoms actually present, and of their mode of extension, will prevent the occurrence of this error, for the clinical aspect of tubercular meningitis is so characteristic that it can scarcely

be mistaken for any other cerebral malady by the experienced observer.

There is, however, another tubercular affection of the cerebrum which is not so easily distinguished from the results of chronic ear disease. I refer to tubercular tumour of the brain. The lesion may be single, but is more often multiple, and I confess that if chronic ear discharge be present I know of no certain means by which it is possible to distinguish this brain affection from cerebral abscess the result of chronic ear disease.

So absolutely alike are the symptoms of the two maladies that I have known trephining operations to have been undertaken on the assumption that abscess in the temporo-sphenoidal lobe or cerebellum was present, itself the result of the chronic otitis media.

This mistake in diagnosis I have observed several times, and, as I said before, I know of no means by which it is possible to avoid it.

A very rare manifestation of tuberculosis is that which affects the tongue. Some years ago a man of thirty-five came to me complaining of a tongue lesion which gave him great pain. He was suffering from advanced phthisis, but there was a clear history of syphilis. On the assumption that the lesion of the tongue was of a syphilitic nature appropriate treatment had been carried out for months; but the tongue became steadily worse.

When I saw him the voice was hoarse, the interior of the larynx reddened, and the left vocal cord ulcerated. The ulcer of the tongue commenced just above the tip of the organ; it spread with great rapidity, and soon became as large as half a crown. The discharge from the ulcer did not contain tubercle bacilli—a point of no importance as concerns the diagnosis. The condition of the patient was extremely unsatisfactory, and no kind of treatment had any effect either on the general malady or on the local tubercular lesion. Death soon followed.

Here is a case in which a tongue lesion, undoubtedly the result of tuberculosis, was thought to be the consequence of syphilis. Had it not been for the rapid development of laryngeal and pulmonary phthisis the diagnosis of syphilis, of which it will be remembered there was unmistakable evidence, would have seemed the only one possible.

These, then, are some of the difficulties and obscurities which surround the diagnosis of tuberculosis. The subject has been illustrated throughout from the records of actual clinical experience, and enough has been written to show how very difficult and misleading the diagnosis of tubercular lesions, as seen by the physician, may be.

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A CLINICAL LECTURE ON LUPUS.

Delivered at St. Bartholomew's Hospital,
December 2nd, 1901.

By J. A. ORMEROD, M.D., F.R.C.P.

GENTLEMEN,—I want to speak to you to-day on lupus. You know that lupus vulgaris is a pretty well-defined disease pathologically, and I suppose I may say also, clinically. Still, you will find there are doubts about certain cases; on the one hand, cases which have received the name lupus, but are not of the same nature as ordinary lupus; and, on the other, cases whereof the diagnosis is doubtful, and which one does not know how to classify, whether as lupus vulgaris or lupus erythematosus, or as neither the one nor the other. It was my intention to-day to show you cases illustrating this point, namely, that there are several doubtful cases, as well as those of which the type is certain, but I do not know how far I shall be able to do so, because my patients have not all come. However, I will do the best I can from the material before me.

By lupus vulgaris, or true lupus, we understand something definite, namely, a primary tuberculosis of the skin. I say primary because we do not include under the name lupus cases where the tubercle of the skin is secondary, spreading from infected glands through the skin to make ulceration or tuberculous lesions on the surface. True lupus is pretty well defined anatomically. It consists of a small-celled growth embedded in a fine reticulum which is in the cutis itself; and in the most typical kinds of lupus this small-celled growth is massed together into little nodules. The skin in which these nodules are embedded is more or less condensed around them. The nodule is not vascular, and, consequently, the cells in its centre are apt to degenerate. In the centre there are occasionally found giant-cells or structures like giant-cells, and in these are sometimes found the tubercle bacilli. So that for all practical purposes,

the nodule of lupus is the same as a miliary tubercle. Secondary changes may take place in the epithelium over it, so that the growth becomes scaly on the surface. But lupus does not always exist in this form, for sometimes, instead of being massed together in definite nodules, the lupous tissue, or the lupoma, as it is sometimes called, is diffuse, and not gathered together into these particular little aggregations. You know that that is just the same sort of thing as may occur in another small-celled growth, namely syphilis. For syphilis may either be massed into definite nodules, gummata, or it may be diffuse, existing in ill-defined areas, as, for instance, in gummatous meningitis.

The clinical characters of lupus vulgaris are as follows:—It generally begins in youth, though it goes on into adult life. It mainly attacks exposed parts, particularly the face, and it may attack the mucous membranes in the neighbourhood of the skin, principally the orifices of the mouth, nostrils, etc. It is generally non-symmetrical, and, strange though it may appear, other manifestations of tubercle are not generally present. For instance, it is rare to find lupus of the skin associated with tubercle of the lungs. Its appearance is most characteristic when the growth is massed into nodules. These appear in the skin as round deposits of variable size, the average size being that of a split pea, and are of a yellowish brown colour, which has been compared to the appearance of apple jelly. They are softer than the surrounding skin. Other secondary deposits form around the primary one, and these are sometimes called "satellite" deposits. It might seem from this description that they are easy to recognise, but the appearance of the nodules may be masked in many ways. The first thing which may so mask the primary lesions is a general hyperæmia of the parts. Where that is the case you can get rid of the hyperæmia temporarily by pressing a piece of glass on to the surface till the hyperæmia disappears and the typical apple-jelly nodules remain visible. In the next place, they may be masked by an overgrowth of the epithelium of the surface, or by eczema with discharge and crusting, or by ulceration of the growth itself. Or, again, the disease may exist, not in nodules, but in the diffuse form. Therefore any patch of chronic persistent inflammation and thickening, with a well-defined

edge, and rather scaly on the surface, if it occurs in a young subject and in a likely part, such as the face, should arouse your suspicion of lupus, and then you should carefully look for any nodules, and if you cannot see them, defer a positive diagnosis until something more happens, such as extension of the growth or scarring or ulceration. Then, too, a diagnosis has often to be made from syphilis, which you know sometimes affects the face, and also scars and ulcerates. The distinction is generally this: that syphilis begins in older subjects, proceeds more rapidly, and there are no apple-jelly nodules, though there may be a certain amount of pigmentation which may counterfeit them. There are also the factors of history and the consideration as to whether or no it yields under antisyphilitic treatment. Another diagnosis which has to be made is from lupus erythematosus, of which I will speak presently, or, again, from chronic eczema, or from erythema, wherein, however, there is no deep ulceration and no scarring. Lastly, other conditions from which lupus may have to be distinguished are rodent ulcer and cancer.

In the woman I now show to you, you will see at once what an enormous destruction there is of the face. The disease began when she was æt. 14½, and gradually spread until one and a half years ago. It has destroyed the skin of the face extensively. There is scarring all round the mouth, and destruction of the cartilages of the nose. There have been ulcers all over the region of the mouth and nose. She is now getting better under treatment. She is being treated by the Röntgen rays, under Dr. Hugh Walsham, and during the time she has been under him she has made good improvement. This is a very extensive case of lupus, and has given rise to a considerable amount of ulceration. The other case which I was going to show you was that of a man who had had a patch of lupus on his arm for twenty years. In him there had been very little ulceration at all, simply a superficial scarring, with deposits of lupous tissue around them. Seeing the two cases together you would scarcely believe that they were really the same disease.

I show you now another case from the electrical department of the hospital. It is true lupus, as I take it, and you will see that, as in the other case, it has destroyed a good deal of tissue around the nose, and has scarred the upper lip, but it has not

gone to the extreme that the other has. It was ulcerated formerly, but since she has been under treatment by means of the Röntgen rays healing has taken place. Good restitution is being made. The patient was only eight years of age when the disease began.

The third case I show you is that of a young woman, who came with a red patch about the size of a shilling on one cheek. At first it looked rather like the result of a sting or some sort of irritation. This could hardly have been its origin, because it had lasted for six months, and was gradually growing bigger. Two of my colleagues diagnosed it as lupus; personally, I feel uncertain as to its nature. It is being treated in the electrical department by a modification of Finsen's light treatment, which Dr. Walsham has adopted for that department. In one sense it is an excellent case for light treatment, because it is limited; it is not ulcerating, and it is very easy to get at. The edge is now less raised, and the whole patch is more diffuse than formerly. It is true that the patch is a little more scaly, but that may only mean that there is some irritation caused by the rays.

At this stage I think I might run over with you some of the varieties of treatment for lupus which have been and are adopted. As the disease is tubercular in origin you would, of course, give the patient such general treatment as might do good to phthisical patients: good feeding, cod-liver oil, and tonics; and many people give, in addition, thyroid extract. Another plan which was formerly tried—I do not know whether to call it local or general—is the injection of tuberculin. This is not so much practised now. There are many forms of local treatment. It generally means, when there are many modes of treatment in vogue, that no one of them is altogether satisfactory. Caustics used to be applied, but I do not think they are used much now alone. For superficial lupus which has not ulcerated and is not spreading fast, a simple form of application is to paint it over frequently with Donovan's solution (consisting of *Liquor Hydrarg. et Arsenici Iodidi*, 1 per cent.). Another form of application is salicylic acid, which may be applied either in the form of a paste with glycerine, or as an ointment, or in what is called a plaster mull, such as has been devised by Unna, of Hamburg, for applying medicaments to the

skin. I show you one of these mulls. It consists of a backing of coarse muslin, within which is a thin layer of gutta-percha on which is spread salicylic acid and creosote. The creosote is put there to stop the pain to which salicylic acid is very apt to give rise in lupus. Another kind of local treatment is scraping, or scraping followed by the application of caustics, or scraping followed by the injection of tuberculin. Then, again, there is the treatment by X-rays, which is applicable to many forms of lupus, and does a great deal of good, and finally, treatment by the light from an electric arc, as advocated by Finsen, of Copenhagen.

I now pass from the consideration of lupus vulgaris to that of lupus erythematosus. The latter is generally considered to be a different sort of disease, although, unfortunately, it bears the same name. Still, several authors give it other names. Unna calls it *ulerythema*, and Malcolm Morris *erythema atrophicans*. However, it is very liable to be confused with true lupus, and it may be difficult sometimes to say which disease you are dealing with. Lupus erythematosus is supposed to be non-tubercular. Tubercle bacilli have never been found in these cases, and inoculation experiments upon animals have not resulted in the production of tuberculosis. Anatomically, lupus erythematosus resembles lupus vulgaris so far that there is a growth of small cells in the cutis, just as there is in lupus, but this growth is never massed into definite nodules, but is diffuse. According to some people, it begins especially in the neighbourhood of the sebaceous glands. This, however, other authorities deny, saying that these glands are only involved secondarily. There are no giant-cells, and no tubercle bacilli. There is an increase of epidermis over it which causes scaliness to appear. And sometimes depending from these scales you see tags which dip down, it is said, into the sebaceous glands. This infiltrating growth degenerates and leaves a scar as it disappears, because the cutis has been involved. But it never breaks down into ulcers.

The clinical characters of the disease are that it begins in middle life, or, at any rate, it does not begin in early youth. It affects, as does lupus, the face, and it also may involve the ears, the scalp, and the fingers and toes, and especially their dorsal surface. Very rarely does it attack the palms of the hands or the soles of the feet, and rarely also

the mucous membranes. It is very often symmetrical, and in that it differs from lupus. The disease is often associated with a sluggish circulation, and when it attacks the fingers and toes it may be mistaken for chilblains. It may begin as plaques, which are congested and infiltrated and well defined and scaly. The patches spread by a gradual pushing outwards of the reddened scaly edge, while scarring takes place at the older parts. But there is no ulceration, and never any of the apple-jelly nodules to be seen. The course is very chronic. The patches may disappear of themselves, but treatment is apt to be unsatisfactory. Quinine is sometimes given, for Dr. Payne says quinine has a power of contracting the capillaries of the face; Dr. Radcliffe Crocker gives salicin for the same reason. Local treatment should not be too vigorous. This patient has had the complaint for ten years. She is now thirty-three. Twenty-three is perhaps rather an early age for lupus erythematosus to commence. Nevertheless the appearance which she presents is fairly characteristic. Perhaps the most diagnostic feature about it is the way in which it is distributed on the bridge of the nose, on each cheek, and on the chin. On the nose it occupies what is called the "flush area," giving rise to the so-called "butterfly patch." Some peculiarities of this case are, first, that it began rather earlier in life than lupus erythematosus generally does; secondly, that there is some reason to suppose that this patient is a tubercular subject, for if you look at her hands you see she has some deep scarring—oddly enough, symmetrical—from which she says bone came away. That is highly suggestive of her having had tubercular disease of the bones there. Thirdly, her face was said to have ulcerated; but, on further inquiry, it turned out that the "ulceration" had only followed on scraping to which she had been subjected. She is now being treated with the modification of the light treatment which I have already referred to. I do not know that that has been generally applied to lupus erythematosus, but this patient came from New Zealand on purpose to be treated by the rays, and therefore it seems only reasonable that they should have a trial. She has only had about a fortnight of the treatment yet, so we could not expect much change.

The next patient I consider also has lupus

erythematosus. Her age is thirty-eight, and she has had the complaint two months. She first noticed it on her face, and then on her ear. It is at an early stage. On her face you see a raised, thickened, reddened, scaly patch, with a very definite margin, and in the centre it is scarring. It is not symmetrical. She had a patch on the ear, which, although it is not so definite in its margins or so scaly, and though there is no scarring about it, is, I think, of the same description. She also has a patch on her occiput. She has been treated with salicin internally, and ointment of zinc ointment has been applied to the patches. The patches have been painted with equal parts of camphor and carbolic acid. The ear has improved, but the patch on the cheek remains very much in its former condition.

Here is a young man *æt.* 25, the duration of whose disease appears to have been nine or ten years. When he came to the skin department a little time ago, he had behind the right ear a reddened, indurated, well defined, scaly patch. It was diagnosed as chronic eczema, but it became apparent on close inspection that it could not be eczema because there was distinct scarring in the middle of it. Indeed there was so much atrophy and scarring that the lobe of the ear was practically destroyed and adherent, and thus it was obvious that it could not be due to simple eczema. I tried all sorts of treatment, but nothing seemed to do him any good, and therefore I took him to Dr. Walsham and asked him to begin the Röntgen-ray treatment. It was applied for some little time, without much benefit, mild applications being tried with the object of causing a slight dermatitis without ulceration. This proved so tedious that the applications were increased in strength, and ulceration was produced. That ulceration has healed, and the condition is as you see it. There still remains a scaly edge, but it is very much less distinct than it was. This man's complaint began rather early in life for lupus erythematosus, yet, on the other hand, there has been no spontaneous ulceration, and no nodules of true lupous tissue can be seen. I am a little in doubt whether to call it true lupus or lupus erythematosus. It may be possibly something different from either.

AN ADDRESS ON STERILITY.

Delivered before the East Sussex Medico-Chirurgical Society.

By **AUGUSTUS W. ADDINSELL, M.B.,
M.R.C.P.,**

Assistant Physician to the London Temperance Hospital.

MR. PRESIDENT,—The subject of sterility has long occupied the attention of workers in gynaecology. Socially it is of importance; professionally it is of interest. In recent years medical science has made great strides in many directions. The bacteriologist is able to determine and demonstrate the micro-organism which generates the poison and causes the terrible spasm of tetanus; we can form a reasonable estimate as to the probabilities of infection of typhoid bacillus long before the classic symptoms show themselves; recent experiments on the antitoxins of diphtheria and typhoid fever, and the extended use of the glandular extracts of thyroid and ovarian tissues, all mark an advance in the domain of science.

But many of the problems connected with ovulation and menstruation remain still unsolved. We are as ignorant as the ancients as to what determines the uterus to expel its contents at the ninth monthly cycle. We have no idea when the ovum leaves the ovary and enters the Fallopian tube. We are not quite sure where impregnation takes place. Formerly it was believed that ovulation and menstruation were coincident. This, however, we now believe to be not necessarily so.

A definition of sterility is not quite so simple as it would at first sight appear to be, and this arises from the fact that there has crept into the literature of the subject the recognition of a difference between relative and absolute sterility. Dr. Gervis, in an able article in Allbutt and Playfair's 'System of Gynaecology,' suggests an elaborate and comprehensive classification; and indeed many subdivisions may be given. But for practical purposes a woman may be considered as absolutely sterile where, living in wedlock during the child-bearing period, she is incapable of conception—most authors add "or of bringing forth a living child," whereas a relatively sterile woman is one who, living in wedlock during the child-bearing period, only bears one or two children. An extreme example of an absolutely sterile woman is where there is absence or ill-development of the uterus, tubes, or ovaries. But

there remain many women absolutely sterile in whom all the organs of reproduction are apparently normal.

With sterility in the male, and there is much clinical evidence to show that it is far commoner than is supposed, we are not concerned; and the manifestly hopeless cases, which are fortunately few, of congenital defect, or malformation, or entire absence of the reproductive organs, such as absence of vagina or uterus, may be regarded as gynaecological curiosities.

The exhaustive work of the late Dr. Matthews Duncan on this subject—his Gulstonian lectures for 1883, and his great experience recorded so fully and so ably, remain to this day the classics of the subject. I offer no apology for constant reference and quotation, and you are all probably familiar with his wonderful compilation of statistics. He estimated that of 4372 women married between the ages of 15 to 44, 3710 had a first living child; 662 marriages were sterile—*i.e.* one in six, or 15 per cent. of all marriages between 15 and 44 were sterile. From his own practice of 504 absolutely sterile women married between the ages of 15 and 45, 337 had been married more than three years.

From Ansell's figures of 6000 cases of married women who had all borne a child, seven-eighths bore one before the end of the second year, the majority before the end of the first year, and he concludes that a woman must not be condemned as absolutely sterile until she has lived in wedlock for four years without giving birth to a child.

All authorities are agreed that the best nubile age of woman is from 20 to 25. The average child-bearing period ceases at 38, whereas menstruation ceases, on the average, at 45 to 50. Thus it will be seen that menstruation and the child-bearing period are not coincident. If the average child-bearing period begins at 25 and ends at 38, we have a period of thirteen years during which a woman is most likely to become pregnant.

It must be remembered that these figures are compiled, and the averages estimated, from hundreds and, indeed, thousands of cases, and although exceptions often occur—and in some instances in my own practice, to be reported later on, have occurred—still the conclusions arrived at by Duncan, Ansell, and other observers are in no way affected by these exceptions, which happen to all of us. Ansell found that in the better classes,

women marrying at 25, living in wedlock during the whole of the child-bearing period, bore an average of six children. Whereas in St. George's-in-the-East, in the poorer classes, women marrying at 25, living in wedlock during the whole of the child-bearing period, produced an average of nine children. Thus, there is a relative sterility in the better classes, and it reaches definite limits in the peerage, where there is an average of six. The cause of this relative sterility is not by any means clear. Dr. Duncan was in the habit of telling those women of the upper classes to live on bread and butter and simpler diet, but I fancy there is another factor at work—a largely increasing tendency towards the limitation of families by the practising of preventive measures. I am certain from personal experience that this practice is extending, and is habitually had recourse to in what is generally called the “better class,” but which I prefer to describe as the more leisured class.

It will be familiar to all that there is a large number of women who only give birth to one child. This type of relative sterility is well recognised. It would appear as if the fertile energies become exhausted, or that child-bearing so affects the general health that recuperation is impossible. I have under my care at present a lady who has never recovered the birth of her child twelve years ago; she is weakly and ill, remains an invalid, yet one cannot discover anything to account for her condition. An interesting instance is a lady who years ago was under the care of Dr. Duncan. She is now forty-three. She married at twenty, was a fine, healthy, strong girl, living in the country and leading a life devoted to sport. She came under my care eleven years ago as a case of sterility, longing and hoping as only a sterile woman can long and hope. She had been married twelve years. After treatment, which I will refer to later on, she became pregnant in three months and was confined of a daughter after thirteen years' sterility, and this completely shattered her health—from being a strong woman she became a wreck—and though very anxious for a son she remained sterile for eight and a half years. I attended her last December with a living son and heir.

To all appearances the fertile energies and the general health were both so exhausted that it took eight and a half years to recover.

Of no one is it truer—and of few is it sadder than of the sterile woman—that “Hope deferred maketh the heart sick.” Which of us cannot call to mind the saddened, tired, hopeless woman who has missed the great longing of her life, and looks with envy upon the poorest woman of her acquaintance, and whom she counts rich, in that she can boast of being a mother?

The ætiology of sterility is so large a subject, and the literature so abundant, that I propose to do little more than enumerate some of the principal causes. No cause can be compared with age in extent and power. Of all the causes which come under our notice, perhaps the one for which advice is most frequently sought is dysmenorrhœa.

The Harveian lectures for 1890, by Dr. Champneys, deal very completely with painful menstruation, and a study of these lectures is worthy anyone's attention.

The cramp-like pains of uterine colic are variously ascribed to some obstruction to the menstrual flow—for example, uterine flexion or cervical stenosis. That acute flexions may cause sterility may be, perhaps, true, that they are the cause of dysmenorrhœa is more than doubtful. The well-known conical cervix and pinhole os is, I believe, always associated with an ill-developed uterus, and often ill-developed ovaries, and in my experience I cannot recall a single instance in which any treatment has been of service so far as curing sterility goes.

In spasmodic dysmenorrhœa the pain felt as the sound passes over the internal os points to a sensitive condition at this point, the true pathology of which is still shrouded in obscurity; so far as my experience goes, it is a noticeable feature in a large proportion of sterile women. With regard to the question of pleasure or desire it is only necessary to say that neither are essential factors in leading to pregnancy; indeed, I know of many cases where there is an active antipathy, and yet pregnancy follows coitus. Spasmodic dysmenorrhœa is usually associated with absence of pleasure and desire, and occasionally with vaginismus. When the dysmenorrhœa is cured, pleasure often is experienced, but by no means always. If vaginismus be present it is necessary to treat this latter symptom. Only those forms of vaginismus, however, which are due to an acutely sensitive fringe of the hymen yield to treatment. The true severer forms, where the

mucous membrane of the vulva when touched causes violent spasms, are extremely troublesome. I have found no cutting operation to equal the application of Paquelin's cautery, and I have had most satisfactory results by this means. The only operative interference for spasmodic dysmenorrhœa I ever use is dilatation by long metal bougies. Of the benefit of dilatation in this disorder there can be no manner of doubt, and it is equally certain that many cases of sterility are cured thereby.

Why dilatation of the cervical canal in spasmodic dysmenorrhœa cures sterility is not at all clear, for in many cases there is no marked narrowing, and the bougies are often readily introduced. Dr. Herman, in his excellent 'Diseases of Women,' puts forward the view that in the sexual orgasm there is a peristaltic movement of all organs formed out of the duct of Muller which helps the coming together in the uterus of ovum and spermatozoa, and Duncan thought that there was dilatation of the Fallopian tube uterine orifices and of the cervix during the orgasm. So far as I know, however, there is no evidence to show that these phenomena occur. We can only accept the fact that sometimes dilatation cures both the dysmenorrhœa and the sterility.

The following may be considered as some of the most usual conditions found to be associated with sterility:

Imperforate, or nearly imperforate, or greatly thickened hymen (*preventing coitus*).

Vaginismus.

Atresia of the vagina.

Conical cervix with pinhole os.

Cervical polypi.

Corporeal polypi with long pedicle.

Pedunculated submucous fibro-myomata.

Acute flexions.

Marked contraction at the external os.

Uterine or cervical endometritis, giving rise to uterine or cervical discharges.

Of these common causes happily most are remediable, and it is with this class that we, as practical men, have to deal. This lecture makes no claim to speculate upon, or solve the mysteries of fertile inadequacy.

There is, however, one ætiological factor in sterility so often overlooked, yet so important that I may be permitted to direct your attention to it. I refer to gonorrhœa. The far-reaching effects of

this disorder are being daily recognised by those who work in the field of gynæcology. At the Chelsea Hospital for Women I have been deeply impressed by the frequency of disease of the appendages due to this cause. It is to be found in the operating theatre, the out-patient department, and the bacteriological laboratory. But it is by no means confined to that class who seek advice at hospitals. I am frequently consulted by women in the more leisured walks of life for sterility; often a careful inquiry elicits a story of infection, sometimes so slight as to pass almost unnoticed, and herein lies the danger, for it is only in the severe forms that a pyosalpinx may result; in many the infection is only sufficient to produce a salpingitis without any accumulation of pus in the tubes, but in either case the closing of the uterine end of the tube by inflammatory hyperplasia is an absolute bar to pregnancy.

Dr. Drummond Robinson, in a paper published in vol. xli 'Transactions of the Obstetrical Society,' points out that collecting the evidence of Berggram, Veillon, and Halle, and his own cases of 117 girls suffering from vulval discharge, in no less than 93, *i. e.* 78 per cent., an organism not to be distinguished from the gonococcus was cultivated, and demonstrated by cover-glass preparations. Moreover, in many of Dr. Robinson's cases, typical symptoms were present; in 39 per cent. there was painful micturition, again in 39 per cent. pruritus vulvæ.

The methods of infection in children of the lower orders is probably due to uncleanly habits and infection of child by the mother with soiled hands, etc.

Quite recently, Neisser of Breslau, the discoverer of the gonococcus, has pointed out that owing to the long persistence of infectiousness, and often to the slightness of the symptoms produced, the sequelæ in women have been under-estimated. He finds that 50 per cent. of women affected by gonorrhœa suffer from serious complications, and he goes on to say that 50 per cent. of involuntary childless marriages are due to gonorrhœa and its sequelæ in men and women. Leir-Ascher in 227 cases of sterility ascribed it to gonorrhœa in 121. Noeggerath in 81 patients with gonorrhœa found 49 absolutely sterile.

Here, then, we have a very potent factor in the causation of sterility, and one which perhaps has not received that attention its prevalence deserves.

In considering this subject, with an earnest desire to arrive at the truth for the sake of the truth, we must not be led into neglecting the fact that there are laws governing fertility over which we can hope to exercise no control, and of many of which we are profoundly ignorant. Whilst fully recognising this, giving it all the weight and consideration it undoubtedly deserves, yet I believe there to be a residuum of sterile women who may by suitable treatment become fertile, though this residuum be a very small one, and having regard to the number of the whole, an almost insignificant minority. To the individuals who go to make up that minority it is a matter of such moment that it becomes manifestly our duty to neglect no means to make ourselves familiar with all the methods which hold out any prospect of bringing about the end to be desired, and though a study of the ætiology of sterility may have convinced us that by far the largest number of sterile women are incurable, yet experience has proved that there are many who by our help may yet find themselves "as women wish to be who love their lords."

I propose to indicate some of those remedies I have found of service in cases occurring in my own practice; perhaps a brief account of some of the most striking will be the best plan to follow.

January, 1895.—Mrs. R—, æt. 27, married five and a half years, menstruated at thirteen, very irregular, and lately profuse. Always painful since age of sixteen, passes clots and shreds, backache, leucorrhœa, vaginismus. Examination was impossible without an anæsthetic as the patient could not tolerate the slightest touch. Under ether the edges of the hymen were destroyed by Paquelin cautery, the cervical canal was dilated with long metal dilators, and uterus curetted; pregnancy followed in four months, and she has since had another child.

January, 1895.—Mrs. B—, æt. 34, married eleven years. Regular, normal quantity, painful; never had connection with her husband on account of the small aperture in a greatly thickened hymen. Under ether: hymen freely incised and edges touched with Paquelin. Cervix dilated to No. 10. Pregnant in three months, has since had two other children.

July, 1889.—Mrs. C—, æt. 32, married twelve years. Regular, painless, profuse, increasingly so last three years; much cervical catarrh with thick

glairy mucus. She had been subject to endless uterine treatment. I dilated and curetted, and glycerine tampons were applied subsequently. In October of the following year she gave birth to a daughter, and her health completely broke down. She remained childless for eight and a half years: uterine and cervical catarrh increased considerably with excessive quantity of glairy mucus: again curetted in January, 1899. Pregnant in April, 1899; confined last Christmas at the age of forty-three.

May, 1896.—Mrs. L—, æt. 34, married eleven years. Menstruated at thirteen, regular and painless up to eighteen months ago, now passing clots and shreds: often laid up for two, sometimes three or four, days at period. Under ether, canal dilated: external os very small, this was incised. Great difficulty was found in passing the internal os, which was eventually dilated sufficiently to pass No. 10. Uterus curetted freely, and many shreds of thickened endometrium removed. Uterus was swabbed out with liniment of iodine on Playfair's probe. She brought forth a living child sixteen months after the operation, and at this moment is again with child. Æt. 38.

1900.—Mrs. L—, æt. 25, married five years. Menstruated at fourteen. Always regular and painless up to eighteen, then had severe chill at a dance. Since then dysmenorrhœa, worse since marriage, increasingly so last two years. There was found to be acute ante flexion, uterine leucorrhœa, cervical catarrh; sound passed half an inch beyond the bend. She was dilated to No. 10 English silver Hagar's, curetted with Martin's curette, and much adenomatous vegetations removed; freely swabbed with tincture of iodine; plugged for twenty-four hours with iodoform gauze moistened in 1 in 4000 per chloride of mercury. She is now six months pregnant.

1898.—Mrs. T—, æt. 39, married eighteen years. Consulted me for an abdominal swelling; hymeneal aperture only admitted a sound. Marriage had never been consummated. Under ether, examination showed thick fleshy hymen, which was incised: large ovarian cyst subsequently removed by laparotomy. The patient became the mother of a child born alive last month. Her age is now forty.

July, 1896.—Mrs. M—, æt. 24, married six years when eighteen years old; menstruated soon after twelve, irregular, painful, and profuse; backache:

sense of bearing down ; been in ill-health for three years past ; dysparunia. Examination showed tender swelling in pouch of Douglas ; acute retroflexion ; prolapsed left ovary. Treatment consisted in replacement of uterus, hot douches ; Schwalbach for six weeks did much good to general health. Her husband went to South Africa for six months, returned in February, 1897, pregnancy followed in May. No uterine treatment was employed.

These serve to illustrate the sort of cases in which interference seems to do good. I offer them as instances where results have followed : in some, at any rate, it may fairly be assumed that without interference sterility would have persisted.

In discussing the various causes of sterility I dealt perhaps more fully with gonorrhœa than with some others which are more usually recognised, and I pointed out that often it escapes our notice because the symptoms are often so slight. It is most usually met with in the newly married ; the husband having contracted gonorrhœa some months prior to marriage undergoes treatment, and is pronounced free to marry—a slight yellow discharge from the woman is almost always disregarded, but if this be submitted to bacteriological examination the detection of the gonococcus reveals its true nature. It is therefore in the early recognition that any hope can be looked for of averting the far-reaching and remote effects, one of which is sterility.

The chief characteristic of the gonococcus is that it is intra-cellular, and it is this which offers the greatest difficulty in its eradication, for infection spreads up the uterus into the Fallopian tubes, and even should it stop short of resulting in a pyosalpinx, the uterine aperture is so minute that the least degree of hyperplasia is sufficient to close it. Our only hope, therefore, lies in arresting the process early, and as soon as detected, dilatation and efficient curettage, with application of antiseptics, such as liniment of iodine, to the uterine cavity should be resorted to with frequent antiseptic douches, and tampons of ichthyol and glycerine. I have had recourse to these measures on several occasions, and have never had occasion to regret it. It may be that in the future, not too far distant, help by antitoxins may be looked for.

All writers on sterility refer to such general conditions as obesity and alcoholism, over-indulgence in the pleasures of the table, heat and cold, and

excess in sexual indulgence. Doubtless these play their several parts, and where there is reason to believe they are of influence, suitable measures are to be advised. Certain it is that healthy surroundings and attention to the general health are points always to be borne in mind. Separation for a time of husband and wife—and where possible, a visit by the woman to such watering places as Woodhall Spa in England, Ems, Schwalbach, and Frinnersbad on the Continent, have in some instances seemed to do good.

One word as to the technique when operative interference is decided upon. Dilatation or curettage should never be done without an anæsthetic ; it is, of course, unnecessary to add that the strictest antiseptic precautions should be observed. It should never be done when there is any pelvic inflammation, for dragging on the uterus with the volsellum is very liable to set up acute inflammatory mischief. It is rarely necessary to dilate the internal os beyond No. 10.

I believe that if there be any laceration of the mucous membrane, as evidenced by bleeding, then the subsequent cicatrisation leaves the canal in a worse state than before interference. The most satisfactory, and certainly the safest curette is Martin's of Berlin. The best time is three or four days after the cessation of the period. I have never seen any ill-results where these precautions have been carried out.

Of the last thirty-three women who have consulted me for sterility no less than twenty have since become pregnant ; of these twenty, the shortest time of married life was three and a half years and the longest nineteen. It is therefore a mere accident that so large a proportion of women have happened to come to me who had a defect which was easily remedied. For instance, no less than six of the thirty had never had connection owing to a thick and impenetrable hymen ; this, by the way, I believe to be far commoner than is generally supposed.

Perhaps the prospect of success in operative interference is not great, but the desire of every woman to become a mother, to prove her womanhood to other women, is very great indeed. In every age, in every country, the cry, the earnest prayer of woman is to bear a child.

Hamlet says, "Conception is a blessing." With the ethics of the question we need not con-

cern ourselves. To my mind it is clear that if by remedying a defect we can convert a sterile into a fertile woman, then our course is plain. I know nothing in life sadder than to just miss a good thing. This pathos of incompleteness in a woman's life who is childless is nowhere better rendered into words than in Paolo and Francesca, when Giovanni says to Lucrezia, "Well! well! you are spared much; children can wring the heart." Lucrezia makes answer, "Spared! To be spared what I was born to have! I am a woman, and this very flesh demands its natural pangs—its rightful throes—and I implore with vehemence these pains."

Blood Poisoning and Amputation.—Heinrich Wolff (*Münch. Med. Woch.*, November 26th) warmly urges the advisability of amputation in blood poisoning, when certain contingencies arise, as follows:—(1) When, notwithstanding ample drainage, the acuteness of the process continues, new tissues being successively involved, and marked systemic symptoms have developed. (2) When the local process remains stationary, but the systemic symptoms are not alleviated, and the life of the patient is threatened by toxæmia or bacteræmia, in spite of the fact that ample antiseptics is employed at the local lesion. (3) When the primary injury of the affected part is extensive, rendering its complete regeneration and subsequent usefulness extremely questionable. Under these conditions amputation is not only logical treatment, but is generally absolutely demanded to save life. Recent bacteriological researches which have demonstrated the rapid absorption (in a few minutes or hours) of the infective agents into the general circulation have led some to suppose that amputation under these conditions is useless, since in spite of this operation a sufficient number of bacteria are thus absorbed to produce death. Such is not usually the case, and in the absence of any specific agent effective against the bacterial infection, amputation is the only logical treatment to pursue. The author reports a number of cases to show that even after general infection has taken place and the presence of the causative micro-organisms in the blood has been demonstrated, amputation is of avail and may save life. In acute infectious osteomyelitis, amputation is indicated, and is often followed by favourable results. A case is reported to emphasise this statement.—*Medical Record*, December 21st.

A LECTURE ON DISINFECTION.

Delivered at the Public Health Laboratory, Guy's Hospital.

By W. C. C. PAKES, D.P.H.Camb.

GENTLEMEN,—I wish to-day to describe to you some of the methods employed in disinfection, and the reasons for these methods. In the first place, perhaps it would be well to define the terms that are in use.

Disinfection means really the actual destruction of any micro-organisms,—which, of course, includes bacteria and their spores,—moulds and their spores, yeasts, and so on, in fact, any form of life, whether animal or vegetable; and a *disinfectant* can be described as a substance or an agent which is capable of actually destroying all the low forms of life. An *antiseptic*, on the other hand, is merely a substance or an agent which prevents the growth of any of these bacteria, moulds, or yeasts. A *deodorant* is a substance or agent which has merely the power of taking up offensive odours. We have, therefore, the three substances or agents—disinfectants, antiseptics, and deodorants.

A disinfectant must of necessity be an antiseptic, but an antiseptic need not be by any means a disinfectant, and a deodorant may or may not be a disinfectant or an antiseptic. As an example of what I mean, if you take a very strong solution, such as 1 in 1000, of perchloride of mercury, and put into this solution a number of bacteria, they will very quickly be killed. This, therefore, is a disinfectant. If, however, you take a very much diluted solution of the same substance, of a strength of 1 in 100,000, all you can say is that the organisms will not multiply in that solution; but experiment shows that they, the bacteria, are not killed in that solution. So that a 1 in 1000 solution of perchloride of mercury is disinfectant, a 1 in 100,000 is antiseptic. Perchloride is not a deodorant at all. On the other hand, a strong solution of potassium permanganate will oxidise the albuminous material, and therefore destroy the bacteria. A weaker solution, although it may not be sufficiently powerful to destroy the bacteria, will prevent them growing, and at the same time will take up any odours that may be given off from the growth to these bacteria; it is therefore deodorant.

In actual practice it is necessary to know something about disinfection and antisepsis, in order that we may, in the first place, prevent the extension of infectious diseases to people other than those who are actually suffering from them, and, as you know, to enable the surgeon to cut down and perform various operations without the danger of infecting the patient with some disease or some bacteria which he has not got. Disinfectants are, then, either substances or agents. One of the best of disinfectants is a high temperature, the temperature imparted by an ordinary Bunsen flame or fire. The application of such a high temperature to any organic substance results in its being *burnt*. The actual combustion or oxidation of the organic matter will necessarily destroy everything in the way of life, whether animal or vegetable, and therefore anything which is of an infective nature, such as the stools of an enteric patient, the skin from patients who are desquamating from scarlet fever, the clothes which have been infected, and so on, will be made absolutely non-infectious by the destruction of the substance which is infected. This is the most satisfactory method of getting rid of bacteria, but, of course, there are various notable objections to it, in that it destroys the material which you wish to disinfect.

Again, we can disinfect by means of *hot air*. If you heat air to 150° C., and place in that hot air bacteria or substances such as the clothes which are infected with bacteria, and leave them in for as long as three quarters of an hour, everything will be killed. Short of this time, and short of this temperature, you can be only sure of killing the bacteria, but you cannot be sure of killing the spores. There are disadvantages as well as advantages about the use of hot air. Hot air has very little penetrating power, and therefore deserves only the name of a surface disinfectant; it is a method which we employ in the laboratory to sterilise glassware and metals.

Another method is by the agency of *steam*. Steam, of course, may be given off at 100° C., and it may be given off at practically any temperature that we like above that. Steam may be either confined or current. *Confined steam* is the steam which is produced in a vessel such as a saucepan, when potatoes are being boiled with the lid on. *Current steam* is the steam which is produced when potatoes are steamed in a vessel with a hole

in the lid. In Koch's steriliser there is a special lid to allow the steam to escape, and it is given off at the ordinary pressure of the air. Steam at 100° C. will, in the course of fifteen to twenty minutes, kill all bacteria. But with current steam at 100° C. it is impossible, under many hours' exposure, to kill the spores of the bacteria, or at all events, the more resistant spores.

We may raise the temperature of steam to produce *steam under pressure*, and it has been found, quite empirically, that steam at 115° or 120° C. is capable, in twenty minutes, of sterilising, *i.e.* disinfecting, any bacteria or spores.

Steam may be either *saturated steam* or *superheated steam*. Saturated steam is steam which is given off from a boiler, or a source of steam at the temperature at which the water is boiling. For instance, the steam coming from a Koch's steriliser at 100° C. is saturated. Steam coming from a boiler which is blowing off at 130° C. is still saturated; the point is that the steam coming off, at whatever temperature it may come off, is saturated when the least amount of cooling is sufficient to ensure the deposition of moisture from it. If steam is coming off from a boiler at 120° C., and it impinges on to a plate at 119° C., moisture will be deposited, and it is therefore said to be saturated. Superheated steam is steam which is given off at some temperature— 100° or 120° C. or more—and is subsequently, by other means, raised to a higher degree than that at which it is given off. If steam coming from a boiler at 120° C. is passed through a chamber which is artificially heated by a Bunsen burner or a fire to many degrees higher, say 300° C., this steam is superheated, and the steam issuing from such a source will not deposit moisture until the temperature has fallen to 119° C. Steam, at whatever temperature it comes off, is vapour, and therefore differs in some respect from an ordinary gas. When steam is superheated, however, it acts more like a gas, and it follows more closely the laws which apply to hot air than those which apply to vapours. We shall see that this has an important bearing on the question of sterilising.

Lastly, heat may be applied in the form of boiling water. Water, at the sea level, boils, that is, gives off steam at the pressure of the atmosphere, at a temperature of 100° C. Actually boiling bacteria in water, even for a few minutes,

is sufficient to destroy them. Steam at 100° C. we have seen does not kill the spores, whereas boiling does; and the reason is probably that the water has the power of penetrating into the dense capsule with which the spores are surrounded, and of thereby bursting the capsule of the spore, allowing the moisture at the high temperature to get inside. Thus, water at 100° C. is able to kill spores, whereas neither steam nor hot air at 100° C. is able to effect this, the probable reason being that the interior of the spore is not heated to 100° C. either by steam or hot air, or that these bodies can stand a temperature of 100° C. provided that it is dry.

Neither intense cold nor great pressure has a disinfectant action.

In addition to the agents of disinfection I have just mentioned, we have various fluids and gases.

With regard to the fluids, we can say that practically all the mineral acids, strong alkalis, essential oils, and many acid salts are disinfectants when used in a strong enough solution. The acids, such as sulphuric, practically have the same action as burning; they take away the water and char the whole of the organic matter, and therefore destroy all forms of life. A strong acid like hydrochloric acid probably converts the albuminous material of which the bacteria are composed into acid albumin, and therefore the whole bacterium is dissolved. Nitric acid, being a very powerful oxidiser, oxidises organic matter, water and carbon dioxide being formed, and the organism disappears. In the same way with the alkalis, the strong alkalis convert albumin into alkali albumin, the bodies therefore disappear and the disinfection is thus complete. In the case of the application of substances like the essential oils, and certain of the acid salts, the bodies are not dissolved; but there is something in the essential oils and in some of the acid salts which renders life absolutely impossible. If we are surrounded by an atmosphere of carbon dioxide, although we do not disappear, life becomes absolutely impossible, so in a similar way bacteria (and therefore spores) when surrounded by essential oils or their vapours, are in such a position that life becomes absolutely impossible.

Certain gases or vapours are distinctly disinfectant; among these are sulphur dioxide, chlorine,

bromine, iodine, formalin—or rather formic aldehyde.

If you take a fluid which contains a solution of an acid salt or a solution of one of the salts of mercury, this will be disinfectant or antiseptic according to (1) the strength of the salt in solution, and (2) the time during which it acts. There is a very popular notion among certain people who do surgical and gynaecological work, that the very mention of carbolic is enough to frighten the bacteria to death. This is by no means the case. Probably all of you have seen—you may have done it yourselves—an instrument dipped into carbolic or perchloride of mercury for a second or two, and withdrawn, under the impression that it was thereby sterile. One has seen such things as scalpels, scissors, and sutures accidentally fall upon the ground, and instead of being discarded, simply allowed to smell of carbolic before being used again. The surgeon has been astonished to find stitch abscesses subsequently develop: the bacteriologist wonders why there are not more stitch abscesses. Perchloride of mercury, carbolic acid, lysol, chinisol, and izal are among the well-known disinfecting fluids, but none of them have the instantaneous disinfecting action which many people seem to imagine. It would be an unnecessary waste of time to give you all the times which these various fluids take to produce disinfection, but I may briefly say that the question as to whether a certain fluid will be disinfectant or antiseptic will depend very largely upon whether spores are present or not. Spores are more resistant to every disinfecting agent than are the bacteria themselves; if staphylococci which do not form spores are placed in quite a dilute solution of carbolic or of perchloride, they are very easily killed. If, however, a cultivation, say of anthrax which has formed spores, be placed in the same fluid, and this is removed (by washing in sterile water), the spores, after they have been immersed for several minutes, or perhaps hours, will be found to multiply when placed in nutrient media, showing, of course, that they have not been killed by the immersion. This means that the fluid was only antiseptic, not disinfectant.

With regard to the gases, *sulphur dioxide*, when perfectly dry, has practically no antiseptic action whatever, but when it is moist it forms, as you know, sulphurous acid, which takes up oxygen

with considerable avidity, forming sulphuric acid, and therefore, by a process of reduction, acts as a powerful disinfectant. *Chlorine* has a great avidity for hydrogen, and therefore acts as an extremely powerful oxidising agent. The same is true of *bromine* and *iodine*. *Formic aldehyde* is a substance which is half-way in the process of oxidation between methyl alcohol and formic acid. Formic aldehyde picks up oxygen with avidity from whatever substance it can, and produces formic acid. Probably part of the disinfectant property of formalin is due to the fact that formic acid is produced. On the other hand, there can be no doubt that the very presence of formalin as a vapour acts like the essential oils, and makes the environment such that it is impossible for life to continue.

DISINFECTION IN PRACTICE.

First of all we will see how a surgeon who is going to operate sterilises himself and the patient. As you know, the skin normally contains large numbers of bacteria, generally, it is true, of a non-pathogenic nature. But sometimes—and of course one never knows when—there are pathogenic bacteria upon the skin. The common organisms are the staphylococcus epidermidis and the staphylococcus albus, which are avirulent, or of very low virulence. Sometimes you find on the skin virulent forms, such as staphylococcus pyogenes aureus. If, therefore, you cut through into the subcutaneous tissue without first cleansing the skin, you may transfer into the tissue some of these organisms, and when the wound is sewn up, they are included in the tissue, and may easily begin to grow and cause the pus formation which is not infrequently seen around stitches. To render the operation safe it becomes necessary to try to sterilise the skin. You know that in the skin there are certain pores and hair follicles in which bacteria may be found. We must admit, therefore, that it is absolutely impossible to sterilise the skin of a patient while he is alive, neither boiling nor burning being feasible. We must therefore be content by removing the largest number possible from the actual surface.

There are various ways of sterilising the skin. The first essential is to get rid of as much of the hard material which forms the top layer of the skin as possible, for it is among this that the bacteria lie. This can be done by thoroughly

scrubbing by means of a nail brush with soft soap, or soft soap which has been rendered alkaline. Lysol is very convenient; it is one of the carbolic derivatives, made distinctly alkaline, and mixed with soft soap. Formerly, liquor potassæ was used to soften the skin, but lysol serves the same purpose, namely, to soften the horny layer of the skin and get away the keratinous material from the surface, and it has the additional advantage of being also a powerful disinfectant. Some surgeons sterilise the skin with perchloride of mercury, and it is sometimes judicious, after scrubbing the skin with lysol and soft soap, to treat it with this salt. One important point to bear in mind, however, is that it is absolutely useless to try and sterilise the skin with perchloride unless every trace of grease has been previously removed from the surface. The perchloride rolls off the greasy skin like water from a duck's back. This is one of the disadvantages of perchloride in sterilising the skin, but it is a very efficient disinfectant after the grease has been removed by alkali. The same procedure should be undertaken in sterilising one's hands. When an operation is about to be performed it is necessary that the operator's clothes should be covered by some garment that is either aseptic or antiseptic. A very useful plan is to have a mackintosh overall, reaching from the neck to the feet, and over this to have a gown of linen which has either been sterilised by being put in some disinfecting apparatus, or by being well wrung out in some such substance as carbolic. Such a procedure isolates the surgeon's clothes from the patient, and then practically the only chance of the surgeon infecting the wound is from his hands, if they are not clean, or from his head, if it is not covered up. In Germany some of the surgeons go as far as to cover up the whole of their head, to prevent chance bacteria from falling into the wound. Although this is not done to any extent in this country it is a wise precaution, especially when the operation is being done on a hot summer's day in a close operating theatre: the perspiration which rolls down the brow is not sterile, and may wash bacteria from the hair into the wound.

With regard to the instruments which are used, as I have said, boiling for five to ten minutes is a very effective way of sterilising anything that can be so sterilised. It is found, however, that the boiling takes away the temper of the instrument. To

reduce this deterioration to the minimum it is advisable to put in a little sodium carbonate to soften the water, so that the water shall not deposit its lime salts. The temper of the instruments certainly keeps better when this is done. Five to ten minutes' boiling in water is sufficient to sterilise any instrument whatever, whereas merely putting the instruments in a 5 per cent. solution of carbolic for two or three minutes before the operation is not sufficient. So long as they are in carbolic it is true that nothing will grow. Supposing you had been operating on a man suffering from erysipelas, or some such infectious disease, and the knife were wiped in the usual way and dipped into carbolic, so long as it is in the carbolic the bacteria will not grow, but directly you make a cut with the knife you smear on the surface of the flesh a certain number of bacteria which have not been killed. The carbolic from the knife is quickly absorbed by the tissues, and the bacteria, no longer being surrounded by the full strength of carbolic, can grow. If, therefore, you boil what instruments can be boiled for five or ten minutes you can use them with absolute impunity. Of course, there are some things used which cannot be boiled; these must be steeped in such substances as carbolic (5 per cent.), lysol (2 per cent.), or other disinfectant, for certainly one or two hours in order to ensure their sterility. Such things as sutures, whether silk or catgut, should be steeped for at least twenty-four hours in a 5 per cent. solution of carbolic, or in a 1 in 100 solution of formalin. When every precaution is taken to prevent bacteria from gaining access into a wound, the operation is said to be an aseptic one. An operation performed under antiseptic precautions is one in which the bacteria are killed by keeping the tissues bathed in a disinfectant, as by using the now old spray. Asepsis is the direct lineal descendant of antiseptics. Lord Lister introduced the method of antiseptic surgery as the result of Pasteur's researches, and I am sure that nobody is more delighted than Lord Lister to see that his child has died of senile decay after having produced such a worthy successor.

Next we want to see what methods must be adopted, and what measures should be taken, to prevent the spread of an infectious disease from patient to patient, from patient to nurse, or from patient to doctor. We have to do with a large number of things to this end. Let us take the case of a

patient who is isolated in a house, suffering from an infectious disease. In such a disease as diphtheria we have to lay special stress on the utensils used by the patient, such as cups, and those which have been put into the mouth of the patient. If we are treating scarlet fever we have to be careful about the skin which desquamates. If the case be one of enteric fever we have to be especially careful about the excreta and anything which is soiled by it. If we take every possible precaution, we may say that we have to deal first of all with the patient himself; secondly with the clothing and bedding; and thirdly, with the room and the furniture in it.

Wherever it is possible to burn we must burn. Therefore, any newspapers and books of no value should be at once burnt. Of course, many things in a room are of value, and it is not necessary to burn them. Articles of clothing and of use, such as handkerchiefs and the like, instead of being burnt, can, as a rule, be boiled. But, of course, it is impossible to either boil or burn the patient himself, at any rate, while he is alive; but there is no doubt that when such a patient is dead the safest way to prevent the disease spreading is to have him cremated. Every year there seem to be fewer objections to cremation, and it seems quite rational that cases of infectious disease, especially where the patients are so dangerously infective as plague, should be cremated after death. But the patient having recovered from the disease, it is necessary to take some precautions in order to sterilise him. In the case of such a disease as diphtheria, it is safe to treat the throat with a gargle of 1 in 1000 of formalin, and to prevent the patient mixing with his friends until the diphtheria bacilli have disappeared from the throat. In the case of scarlet fever you have to take care that the patient has absolutely completed the process of desquamation before he is allowed to mix with his fellows. In enteric fever, as we know, the urine may be a source of danger, even well into, and sometimes after, the convalescent stage. The fæces may remain infected for some considerable period after convalescence has set in; and therefore the fæces, both during the disease and after the convalescence has set in, must be specially treated. One of the methods of treating the fæces and the urine is to disinfect them with one of the disinfectant fluids. This is the way generally adopted in

this country, and if it is done properly there is no objection to it. There are two fluids chiefly used for the purpose, perchloride of mercury and carbolic. The one great objection to perchloride for the disinfection of the evacuations is that there is an enormous quantity of albuminous material present in the latter, and when a solution of perchloride is added to it a precipitate of albuminate of mercury is formed, which is not disinfectant. In order to guard against this, care must be taken to have a considerable excess of perchloride dissolved in water containing free hydrochloric acid. The perchloride should be in a strength of 1 in 1000 after mixing with the stools. A similar objection holds in the case of carbolic, but not to the same extent, and the carbolic after mixing should be in a strength of 1 in 20. Such solutions left in close contact with the stools for upwards of one hour, will probably kill all the pathogenic organisms, and the excreta can safely be thrown down the drain.

Certain special apparatus has been invented in order to burn the fæces. There is no doubt that this is very effective, and the best way of getting rid of them; if this were done one would never get the water-supplies contaminated by the fæces of patients who are suffering from such diseases as enteric fever. In the present South African campaign the stools were boiled in a copper in the open air, and this method might well be adopted in country districts.

In speaking of clothing, I said all that could be boiled should be so treated. But there is one objection to this, namely, that stains remain on the article when they are put directly into boiling water. To overcome this difficulty the articles before boiling may be soaked for some time in cold carbolic ($2\frac{1}{2}$ per cent.) until the stains come out. They may then be boiled without remaining stained.

One of the most important items we have to consider is the bedding. If you have had a patient with diarrhœa, from enteric fever, for instance, and he has passed his evacuations underneath him, the thick mattress gets infected with the bacteria and is a source of danger. If the mattress is a cheap one it may be advisable to burn it. In the case of poor patients, sanitary authorities often prefer to burn the ordinary cheap straw mattress and provide the patient with another, rather than go to the expense and trouble of disinfecting it by other means. But sometimes the mattresses are not so

cheap, and then it becomes worth while to disinfect them. And here comes the difficulty, because we must be sure that the disinfection is complete, and we require some means of reaching both the inside and the outside of the article. Hot air has very little penetrative power, and cannot therefore be employed. Steam at 100° C. is useless, because it does not kill spores which may be present. The alternative is to disinfect with steam under pressure at 120° C. If the mattress is kept at that temperature for twenty minutes it will be disinfected. It is necessary that the interior as well as the exterior should be heated to that temperature. If superheated steam is used the penetration will not be as great as if saturated steam is used. In order that the steam should penetrate properly various appliances are used. These consist of iron jackets of large size. When the mattresses, blankets, and other heavy articles of bedding and clothing have been placed in the machine, and this has been securely fastened, as much of the air as possible must be withdrawn; this is done by a steam exhaust. After being exhausted the steam is allowed to enter, and it penetrates the interstices of the materials. By turning on the steam and vacuum alternately penetration can be assured. After sterilisation is complete, the steam is exhausted and hot, dry air is allowed to enter the machine. This is exhausted and more hot air allowed to enter. In this way the vapour in the bedding evaporates, and the articles may be removed from the machine virtually dry.

With regard to the furniture in the room itself, rooms in this country are generally disinfected by burning sulphur. This produces sulphur dioxide, and, as such, is practically useless. But when combined with steam given off from a kettle, sulphurous acid is produced, and this is a good surface disinfectant. The penetrating power is, however, small. Chlorine is another gas used, but the great disadvantage of this is, that although chlorine is a powerful disinfectant, it is also a powerful bleacher, and if it is used where such articles as coloured curtains are in the room, they will be effectually bleached. In Austria and France formalin is largely used, either by spraying on the formalin by an actual spray or by mopping the floor, ceilings, and walls with the fluid by a brush. This is painful for the disinfectant, as formalin vapour is very irritating to the eyes. The

formalin may be generated outside the room and forced in. After the formalin has been allowed to remain for some length of time the door is opened and the vapour is neutralised with ammonia; three or four hours after this it is possible to go into the room with safety and clean it up. Formalin may be generated in three ways. First by passing the vapour of methyl alcohol (which is heated in a vessel) over red-hot platinum. This method is unsatisfactory. Secondly, by driving off the vapour from a solution at 130° or 120° C. in a machine. Thirdly, by heating para-formalin, a modification of formalin which is solid, which volatilises as formalin. The great objection to all these methods of room disinfection, even with formalin, is that the degree of penetration is only small, and it has been found that organisms which are put two or three inches inside mattresses are not really killed, and even when a room has been treated with formalin for twelve to twenty-four hours, thorough disinfection may not have been carried out. My opinion is that the best way to disinfect a room is first of all to spray it with formalin, then to strip the walls and ceilings and floors, and wash the whole place with such a disinfectant as carbolic. That is a very radical method, but if you want to do it thoroughly that is the best way.

Furniture is very difficult to deal with, and in that matter there is no doubt we are between the devil and the deep sea. If you thoroughly disinfect the furniture you will probably spoil it, and if you keep it from being spoilt the probability is you will not disinfect it properly. It is needless to say what a variety of articles of furniture there may be in a room, even when you have stripped it of encumbrances in the way of upholstered furniture. But there are one or two points with regard to sterilising furs and other similar articles which would be spoilt by being subjected to steam. One of the best ways of sterilising books and furs is to put them into a formalin chamber. Formalin has got rather a bad name because things have sometimes been found not to be sterilised. I think the reason why so many surgeons have accidents after sterilising, and why articles are found not to have been sufficiently treated, is that half an hour's immersion is not long enough. In some experiments in connection with anthrax which I made some years ago, I

found I could not kill the spores of anthrax, even those on the surface, with formalin vapour, in a less time than ninety hours' immersion. The majority of people think twelve hours' subjection to the formalin is enough to kill everything, but you will see that you cannot be quite sure of sterilising even books under ninety hours. It is important to remember in the case of sterilising books, to open the leaves thoroughly to enable the formalin to get into every part, as by hanging the book up with the leaves open. I cannot too strongly insist that the sterilisation should be carried out for many hours.

Omental Fixation for Relief of Ascites from Hepatic Cirrhosis.

—The following anastomoses have been found in dogs by injection by Kusnetzow (quoted in the *Boston Medical and Surgical Journal*, May 16th, 1901):—Numerous ones between the veins of the stomach wall which empty into the superior gastric vein, and those which empty into the large vein along the greater curvature; a direct connection between the vena gastroduodenalis and duodenojejunalis, and an anastomosis between these two and the portal vein trunk through the pancreatic veins; anastomoses between the neighbouring veins, which on one side open into the large and on the other side into the small mesenteric veins.

These anastomoses permit the passage of the blood in case of ligature of the portal vein from the part below to that above a ligature of the portal vein.

Kusnetzow is convinced that ascites due to impeded portal circulation may be treated with excellent results by omental fixing. Without previous omental fixing, it has been found that complete ligation of the portal vein rapidly causes death, and that omental fixing enables animals to bear a double ligation of the portal vein—a complete one in its middle part (above the entrance of the gastrolienalis), and an incomplete one near its entrance to the liver. Complete ligating below the vena gastrolienalis caused death in a few hours even after previous omental fixing. The incomplete ligating of the portal vein in its middle part caused stasis, ascites, and bloody diarrhoea that was transitory. Kusnetzow also found that the superficial epigastric veins dilate markedly after omental fixing, resembling the "caput medusæ" seen with hepatic cirrhosis.—*Therapeutic Gazette*, December 15th, 1901.

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A CLINICAL LECTURE

ON A FEW CASES OF

EARLY CEREBRAL SYPHILIS.

Delivered at Charing Cross Hospital Post-Graduate
Course, November 28th, 1901.

By F. W. MOTT, M.D., F.R.S.

GENTLEMEN,—Some cases of not frequent occurrence having come into the hospital, I thought I would take the opportunity of delivering a lecture upon them, because they illustrate a subject in which I am particularly interested, namely, cerebral syphilis. Some time ago I published sixty cases of cerebral syphilis, and among these sixty there were forty cases in which I was able to obtain a pretty reliable history of the period of infection and the time that elapsed afterwards before the onset of the cerebral symptoms. It is known, but I think it is not sufficiently known, that cerebral syphilis frequently comes on very soon after infection, in fact, the greater number of cases occur within the first few years of infection. Among these forty cases there were five in which cerebral symptoms occurred within the first twelve months, seven in the second year, six in the third year, and three in the fourth year. That is to say, twenty-one cases out of the forty occurred within the first four years; the remainder occurred at successive periods afterwards up to twenty-five years after infection.

CASE 1.—Now there happens to be in this room a young man, B. A—, æt. 22, who was infected with syphilis, but it was not a typical hard sore that he had. For some time he was treated with ordinary local applications of iodoform. Within three months of his having the sore he began to complain of headache and stiffness of the neck. He was seen at the hospital here by Dr. Clogg, and was immediately put upon mercurial pills. He was infected in March, and in July he began to take mercury pills, because that was the time he began to have headache and stiffness in the neck. He continued with the pills, but at the

end of October he left the pills off and was given some medicine—probably iodide. On October 23rd he began to see double, so that he had already, three months after infection, symptoms of meningeal irritation in the fact that he had headache and stiffness in the neck, but other definite symptoms were wanting. It is possible that if the temperature had been taken for two or three days at that time it would have been found that he had an irregular pyrexia, but not necessarily. Lang has pointed out that even within a very short time of infection you may have headache, irregular pyrexia, double vision (from squint), and affections of pulse, that is to say, slowness or increased frequency. This authoritative statement supports the probability that this patient had, within three months of infection, a syphilitic inflammation of the meninges. You see that for three months he apparently had had no internal treatment, so that the poison had had time to work its effect. In October, as I have said, he began to see double, and he had severe headache and weakness of the right arm and leg, slight paresis of the right side of the face, the tongue deviating somewhat to the right, and dilated pupils. The reflexes were slightly exaggerated on both sides, more on the right than on the left. He suffered from very severe headache, which became worse at night. This patient had double optic neuritis, not very marked. By putting him on large doses of iodide of potassium and using mercurial inunction he has greatly improved; strength has returned to his limbs, the squint has disappeared, and he is altogether very much better. That is one case in which the symptoms came on within three months of the infection (probably) and certainly within six months he had obvious and obtrusive symptoms of cerebral disease. I have no doubt that under treatment he will make a very good recovery, because he has been seen early, and he has been thoroughly treated.

CASE 2.—W. R—, æt. 46. I have another case to show you, whom some of you may have seen eight years ago at this hospital. At the time of the onset of his illness he was a commercial traveller in Japan. He contracted venereal disease; it was considered to be a soft sore, and was only treated locally. Six months after he had the infection he was seated at the piano—playing and singing—when all of a sudden

he felt faint. He got up to get some water, and the water came out of his mouth and through his nose. He could not swallow it, and was unable to speak to tell his friends what was the matter with him. They, at first, regarded the matter lightly, but soon saw that he was very ill, and put him to bed. In the morning he was seen by a Dutch physician, who recognised the nature of the disease, and put him immediately upon mercurial inunction. This attack was due probably to thrombosis of a small artery supplying the bulb. Probably the thrombosis extended and affected the pyramidal system of fibres on both sides to some extent, but more particularly the left side, because he shortly became paralysed on the left side. He continued under treatment to improve, and recovered the power of swallowing and speech. But even now speech is somewhat affected. Still, he has made a very good recovery considering the serious nature of the lesion. You will notice some slowness and difficulty of articulation, and that his tongue still protrudes a little to the right side, but he is able to do his work, and to earn his living as a clerk. At one time he could not use the muscles of his forehead on the left side to frown, but he can now. He has a somewhat exalted expression when he enters into conversation, owing to the loss of control of emotional mobility of the facial muscles, and you can also notice a little late spasm of the left side of the face. The paresis was on the left side, and he had weakness of the soft palate on that side. The left vocal cord was affected at the time, but bilateral association allowed a complete recovery. He had exaggerated knee-jerks on both sides, and there was a wrist-tap contraction and ankle-clonus.

The other patient sitting here is one which I will refer to presently. I want first of all to speak of certain cases which I have not here for your inspection, but which have come under my notice at Charing Cross Hospital or at the asylums. These are three other cases, which are examples of early cerebral syphilis.

CASE 3.—R. H—, stableman, æt. 29, contracted syphilis in July, 1897. He attended Guy's Hospital for several months. He suffered with secondary symptoms, rash, and sore throat, and when they disappeared he thought himself cured, and never went any more. That is often the history. Fresh throat trouble commenced in January, 1898,

and in February Dr. Maude was called in to see him. He found that the patient had fronto-temporal headache on the right side, and some tenderness to pressure, and an irregular pyrexia. The pulse was unusually slow, the young man was feeling ill, and the headache was worse at night. He had no squint or vomiting. Basic meningitis was diagnosed, and for some months he was kept upon mercury and iodide of potassium. In the beginning of August, 1898, he complained of numbness in the left hand and arm, then a few days later, almost suddenly, he fell down in a fit, but did not lose consciousness completely—he was merely dazed. This left him with left hemiplegia and dysarthria, for which he was admitted to Charing Cross Hospital. When he came in he had a left-sided hemiplegia and double vision; the headache was very severe indeed, but there was no vomiting. He had slight optic neuritis. Under treatment he did not improve at first, although large doses of iodide were given; in fact, a new symptom developed, namely, very severe pain in the limbs, particularly the left arm. Superficial reflexes present; knee-jerks absent. Muscles flabby and wasted. All the muscles reacted normally, however, to galvanism and faradism except the interossei of the left hand. He complained of stiffness in the neck and back. His mental condition was one of slight dementia, marked by slowness of thought and comprehension. He did not improve much for some time, and the pains in his limbs were so severe as to require morphia injections. I diagnosed syphilitic endarteritis with softening of the motor region of the right hemisphere and cerebro-spinal meningitis. Under continued antisyphilitic treatment he made a partial recovery. He has still left-sided paresis, and there is considerable enfeeblement, but he is able to earn his living as a gardener. I kept him under treatment for two years.

The slowness of thought and comprehension in this case showed that a considerable portion of his brain was affected, no doubt due to disease of arteries as well as of the membranes. In fact, an examination of the brains of many of these cases shows that it is very seldom you get a case of pure meningitis without endarteritis and peri-arteritis as well. These latter may not cause obtrusive symptoms unless the lumen of the vessel be blocked, and that may cause a fit, with sudden or partial

loss of consciousness. If the vessels affected are situated in the region of the brain connected with the higher functions of the mind, and not with the motor functions, or with some sensory functions, then for a long time the disease may be going on without any very definite symptoms. Those are the dangerous cases, and they are the ones which come into the asylums diagnosed as general paralysis, because there is a progressive dementia. But there is a character about such a case which generally leads you to suspect that it is syphilitic brain disease and not general paralysis.

I have in mind two cases of that sort.* Of course there are many in the asylums, but these were of special interest because the cerebral symptoms came on within one year of infection.

CASE 4.—E. J—, æt. 36, carman, admitted to Hanwell, July, 1897, for dementia. Contracted syphilis eighteen months ago. Secondary symptoms, sore throat and rash, for which he was treated at the Lock Hospital. He has never suffered with headache. Twelve months ago the first symptoms of brain syphilis occurred; this was drooping of the eyelid, *ptosis*, although he had been taking mercury pills for six months.

Six months later he had a fit, followed by right hemiplegia, aphasia, and dementia. While in the asylum he has improved a little under treatment, but his mental condition is so much impaired that it is probable he will never recover sufficiently to be discharged.

CASE 5.—W. R—, æt. 26, occupation painter, contracted syphilis February, 1893. In the following October he suffered with partial left hemiplegia. In January, 1894, he came under Dr. Galloway at the Great Northern Hospital, and antisyphilitic treatment was adopted, but not continuously. In the following August he had another severe fit, in which he lost consciousness for one hour. Optic neuritis, which had been present for some months, was passing into post-neuritic atrophy, and he left the hospital. In October he was found by the police wandering at large, and was admitted to Hanwell.

The principal symptoms were ptosis and oculomotor paralysis of right eye, optic atrophy and blindness, tremor of tongue and face muscles, epileptiform convulsions, constant and severe

* These, amongst others, are described in the 'Archives of Neurology,' Pathological Laboratory, Claybury, vol. i.

headache, mental confusion, incoherence, loss of comprehension, and of orientation of time and space. The dementia progressed, and he died four months after admission, or two years after he had contracted the syphilis. At the autopsy we found six or eight large gummatous masses within the brain, besides basic meningitis, peri-arteritis, and endarteritis.

These five cases are not only of interest because they show that severe syphilitic brain disease can arise within three to eight months after infection, but also because they show that there is no relation between the severity of the initial symptoms and the character of the sore to the serious nervous disease which may ensue.

Cases 1 and 2 were both believed to be soft sores, and only received local treatment. There is another disease, namely, diphtheria, in which the throat symptoms may even be so mild as to escape notice, and yet be followed by a severe form of diphtheritic paralysis. Hitzig and Buzard believe that there may be several toxic agents in syphilitic affections, and in cases which are followed by nervous symptoms a special poison may exist which acts upon the nervous system. There is some basis for this hypothesis in a series of cases in which severe nervous disease followed infection from one and the same source, as it is estimated by Hjelmman that only 2 per cent. of the people infected with syphilis suffer afterwards from brain or cord disease, excluding the parasyphilitic degenerative processes, general paralysis, and tabes dorsalis.

The two latter diseases are much more likely, it seems, to follow mild attacks of syphilis than the true specific inflammatory lesions. At one time I thought that cases which were treated from the beginning with mercury would almost certainly escape serious nervous sequelæ, but, as you will notice, both Cases 3 and 4 were under anti-syphilitic treatment for a considerable time, and yet developed severe general brain disease, leaving them paralysed and partially incapacitated, and I have one case under observation now suffering from both spinal and cerebral symptoms, who was under treatment for three years at the Lock Hospital.

The explanation is possibly in some of these cases that the nervous system is the "locus minoris resistentiæ" of the individual.

The early diagnosis, however, of these cases is of very great importance, because of all the serious organic diseases of the nervous system they are the most amenable to treatment, and it is quite wonderful to see what improvement they will make when treated with large doses of iodide of potassium and mercurial inunction. These five cases illustrate, moreover, the different types of syphilitic affection of the brain.

It is seldom that the types are pure and distinct; oftener the disease is a combination, but in Case 1 we had probably to do with only a basic meningitis. Case 2 was probably a simple endarteritis of a bulbar artery, with thrombosis of one of its small branches, causing necrotic softening of cranial nuclei on one side, and involving the pyramidal tract. Case 3 began with basic meningitis, and was followed by extension of the same into the right sylvian fissure, with endarteritis and peri-arteritis of the middle cerebral artery, causing left-sided hemiplegia and slight optic neuritis, with the extension of the disease to the spinal membranes.

Case 4 illustrates the same condition, but the mental impairment, terminating in dementia, indicated a greater extent of damage to the brain; but the fact that there was a right-sided hemiplegia would show that the left hemisphere was the seat of extensive disease, and the associated aphasia would make the mental trouble more apparent, and less capable of recovery.

Case 5 was one of those very severe cases of brain syphilis which terminate fatally, and which are sometimes termed pseudo-general paralysis. Here was found not only basic meningitis, but also general arteritis and peri-arteritis, with multiple gummatous tumours. Possibly if this man had been treated systematically from the beginning with mercury and large doses of iodide of potassium later, he might not have developed this severest form of syphilitic brain disease, which terminated fatally two years after infection. These cases illustrate also the fact, which the sixty cases that I collected show, that syphilis never strikes the nervous system without warning. Too often the warning is neglected by the patient, and sometimes by the doctor, and it is these warning symptoms which I would especially call your attention to. Fortunate is the patient who sees double, or has a droop of his eyelid; it will take him probably

to the doctor or the ophthalmic hospital, and a history of syphilis will very probably be obtained, for it is by far the commonest symptom. It is so common, indeed, in brain syphilis that Fournier and Ricord speak of it as "la signature de la vérole." Usually it is the motor oculi which is affected. Several of these cases exhibit this symptom.

Again, visual troubles from affection of the optic nerve or chiasma, resulting in blindness, may bring the patient. An earlier and more constant symptom still is headache. It may even indeed be the only symptom for some time; the pain may be general or local, dull, aching, or stabbing, generally with paroxysmal exacerbations, usually worse at night, even waking the patient at a particular hour. There may be local tenderness on pressure if the convexity of the hemisphere is affected, but when it is the base of the brain the pain is deep-seated, and referred to the frontal, temporal, parietal, or occipital regions, and it cannot be definitely localised. The headache may arise from inflammation of the meninges, from increased intracranial pressure, caused by gummatous tumours, or simply from endarteritis. Vomiting and vertigo may occur, and stiffness of the neck is one of the early symptoms of the meningeal affection. Optic neuritis may come on early, as in Case 1, or it may be absent all through. It may be due to spread of the inflammation to the optic nerves, as is probably the case in the first patient. If you examine his discs you will find they are red and swollen on both sides, the margins indistinct, but not striated. There is a diversity of opinion about the temperature in syphilitic meningitis; in some cases there may be an irregular remittent pyrexia, as in Case 2, but generally speaking, pyrexia is against the meningitis being of syphilitic origin, unless other indications point definitely that way.

Charcot pointed out that transitory attacks of aphasia, word-deafness, and word-blindness or hemiparesis were common symptoms of syphilitic disease of the arteries. These attacks may last a few hours or a few days, and then, as collateral circulation is restored, there is recovery of function. In connection with this, I may mention that there is another case, which I will show you, illustrating this point, but I should state that there is another and more serious disease—general paralysis—the out-

come of syphilis, which likewise is attended by this phenomenon. Such warning attacks should never be neglected; the patient should be immediately treated in order to prevent the extension of the disease, or what will happen is this: the endarteritis which has occasioned the symptom will extend, and the blood will clot in one of the large branches of the artery, or in the main artery itself, the collateral circulation will be impossible, necrotic softening of the portion of brain supplied by the artery or one of its large branches will take place, and a permanent loss of function will ensue, as in Case 4, producing permanent hemiplegia and aphasia.

Epileptiform fits, general or Jacksonian in character, may be the first indication of the brain disease, especially when there is a gumma situated in the Rolandic region of the cortex.

Mental symptoms.—Disturbances of consciousness play an important part in the symptomatology of brain syphilis. Recurrent attacks of drowsiness, stupor, and coma should always make one suspect, in a young male adult, syphilitic meningitis, especially if combined with one of the symptoms previously mentioned. Dementia is a common result, and due in part to disturbances in the circulation brought about by general endarteritis. A characteristic feature is the variability of the extent of the dementia. Sometimes there is an alteration of character, *e.g.* the genial man becomes morose and taciturn; sometimes there is delirium, with maniacal excitement and motile restlessness, and sometimes extreme mental depression, with delusions of persecution and attempt at suicide. Not infrequently, as in Case 5, the patient is found wandering at large by the police. Extensive destructive changes may occur *in silent parts* of the brain without producing well-defined symptoms, and it is only some act which denotes that they are incapable of taking care of themselves which brings such patients under medical observation.

I will now show you the case of transitory word-deafness and amnesia.

CASE 6.—A. B.—, occupation policeman, æt. 38, admitted to Charing Cross Hospital November 4th, 1901. He complains of pain in the left side of the head. The family history is that his wife has had six pregnancies; one miscarriage at about three months; second child, a girl, living, æt. 14 or 15, quite healthy; third a miscarriage; fourth

and fifth living children ; sixth, a baby prematurely born at seven months, since dead. All the children are said to have had snuffles in infancy. This man had syphilis about eighteen years ago, and that probably accounts for the symptoms which came on. He was under a chemist for twelve months, and then he went to a private doctor, who gave him medicine. There is nothing particular in the man's past history beyond that, except a history of an injury to his head. He was run over twelve months ago. His present illness is as follows, first from the notes supplied by the medical practitioner in attendance :—On November 3rd.—The patient performed his duty yesterday as usual, without complaint. This morning he paraded as usual at 10 a.m., and it was not until 11.15 that any peculiarity was noticed by his superior officer in conversation and conduct. His speech was then noticed to be rambling and incoherent, and he was taken to the doctor. When examined by him in the forenoon he was found to be suffering from pain in the region of the left ear. The present condition reads as follows :—The patient was admitted to Charing Cross Hospital November 4th, at 1 p.m., speech irrational, answered some questions correctly, but answers to others rather far from the point. Seems to feel pain over the left ear and frontal regions ; sometimes seems to have pain and tenderness above and behind the left ear, but that pain is not constant. A plug of wax was removed from the ear, and boracic fomentations were applied. His temperature 100.6°. Patient passed a good night. Evening temperature 101.4°, now 100°. Chest and heart normal. No loss of power in legs and arms. Slight paresis of the right side of the face. The plantar reflex and knee-jerk both well marked, better on the right than on the left. Type of plantar reflex normal. No abnormal reflex in arm. Visceral control normal. No squint ; no ophthalmoscopic changes on either side. Pupil reactions normal ; pupils equal. The patient seems to be deaf on the left side, but this is difficult to be certain about on account of the mental condition of the patient. Speech and motor functions normal. If asked a question he does not seem to understand, and certainly cannot answer correctly, nearly every question receiving the answer, "There is nothing much to show for it ; there is not much to see." Mr. Boyd told me that he could not repeat sen-

tences, and seemed to have word-deafness and loss of memory for words also. But he could read aloud and remember words ; therefore his visual word-centre was all right. That localised the lesion to the upper end of the first temporal convolution of the left hemisphere, and that accounts for the slight right facial paresis, because of the proximity of the motor part of the ascending frontal, where the face centre is. The patient's idea of the meaning of what he had read did not seem to be correct. He can count fingers held up before him, so his sight was all right. When I saw him he was able to repeat after me a sentence, and he was better. There was no difference in the pupils ; there was tenderness and a little redness behind the ear. When he came in the idea was that he had had an injury—some injury to the brain—which might necessitate an operation, or that he had ear disease, which also might require operation. His head was already shaved ; therefore an operation was clearly contemplated. But the symptoms were not sufficiently definite to warrant it, and it is fortunate, in view of what we know now, that it was not done. When I saw the patient I suggested the possibility of specific trouble, and no doubt Mr. Boyd was of the same opinion too, although one could not get a proper history from the patient on account of his mental state. There was definite tenderness and redness over the ear, and that looked rather like there being some local disease present. He also had pain at the back of the eyes. He has weakness of the left leg, and of both feet. Very often that is the history which you get in specific brain disease ; a transitory, sensory, or motor aphasia lasting a few hours, or it may be days or even longer, then the patient recovers from that because, as the collateral circulation comes back, the part is restored to functional activity. But there is another disease in which transitory aphasia sometimes comes on, and that is general paralysis, which is a much more serious condition, and it is no good treating it with iodides and mercury, for, as a rule, they do no good at all. I will now ask the patient to leave us while we further discuss his case. His speech does not suggest general paralysis, and he had pupil reaction to light. In cerebral syphilis, if the pupils are affected they are very often unequal. This man's pupils are a little unequal, the left being larger than the right ; I will test the pupils

again. I do not think they now show any reaction to light. In cerebral syphilis it is more often that the pupils do not react either to light or to accommodation, and you frequently get an ocular paralysis as well, whereas in dementia paralytica you find the Argyll-Robertson pupil present. In quite 50 per cent. of paralytic dementia cases you get the Argyll-Robertson phenomenon in the pupil. There is another disease in which you find it even more strikingly and frequently, and that is in tabes dorsalis. All general paralytics are of this type. Remember that the man whom I have been speaking about, the ex-policeman, is thirty-eight, just about the age at which general paralysis is liable to come on. You notice that he has a very much exaggerated knee-jerk; there is no tremor of the lips or tongue, and his speech is not hesitant, tremulous, or syllabic, which symptoms are among the early indications of general paresis. The man shows no signs of early dementia, or any noticeable alteration in his character. His memory is pretty good. In spite of the absence of these symptoms, I do not regard the case as particularly hopeful, but I shall not give the patient an indication of my state of mind in that respect, because I have often seen incalculable mischief caused by telling the patient that his case is hopeless. It is most important to tell these patients not to worry, and more especially not to drink. As a matter of fact, I regard the failure of the pupil to react to light as of very great significance indeed. Moreover, I have seen so many patients in which there is a history of a fit similar to this many years before, who have later on developed general paralysis, that in spite of his apparent recovery I should regard the case as serious.

The case of the man I showed you first, who was a traveller in Japan, is in reality very much better, although his case was apparently very much worse. His was a coarse lesion, which produced serious damage, and left its effect, but it is non-progressive. But in a case such as that ex-policeman presents, one can never know what is going on. His age and other circumstances would make you give a very bad prognosis in his case. We have not yet determined whether there is any neuropathic history in his family. Of all the causes of general paralysis undoubtedly syphilis is by far the most important, and the more I work at the subject the more I feel inclined to think that

without syphilis there would be no general paralysis and no tabes. But you will find in the majority of these cases of general paralysis that either neurosis, insanity, or potential insanity existed in the family. By potential insanity I mean that the man comes from a nervous stock, and if a poison entered the system of any member of that stock, or some other calamity happened to excite the brain, then such a person might become insane.

However, to return to brain syphilis and its prognosis. Cases of transitory aphasia, which are due to syphilitic disease, if they are seen early are generally satisfactory as regards treatment; that is to say, if you have a man who loses speech for an hour or two, or a day or two, and you can treat him in that early stage, he will generally make a good recovery. Taking all cases of brain syphilis, it has been said that about a quarter recover completely, a quarter recover with some defect like this man W. R—, who can still lead a useful life; and the remainder become either permanently incapacitated, mentally or physically, or both, or they die. So I think both these patients, Cases 1 and 2, have reason to be satisfied with their condition, and especially the latter, because eight years have elapsed since the active disease. It is remarkable how well some of these cases do. There has been a woman in the wards of this hospital whom I would like to have shown you, but she is now gone out; she had all the symptoms of cerebral tumour—vomiting, headache, paresis of the left side, and alteration of pulse. We put her on large doses of iodide of potassium, and she was dismissed practically cured. I should like to mention that I think it is always well to adopt energetic treatment at once. I give them mercurial inunction, generally a drachm Unguentum Hydrarg. with a drachm of Lanoline, and have that rubbed in night and morning until the gums get spongy. But you must always remember that the gums will often get spongy before the patient gets thoroughly under the influence of the mercury unless you insist on him keeping his teeth clean, cleaning them after every meal, and using a mouth wash of chlorate of potash. You may often find you are stopping your mercury on account of sponginess of the gums before the patient is properly under its influence. In the next place, it is of no use to give small doses of iodide of potassium; it is better to begin with 30 grs.,

three times a day, right off. You can even give larger doses than that, you need not be afraid, because you are more liable to get iodism if you give the drug in small doses than if you give it in large ones, and in diseases of this sort you have not time to wait. After a time, when the patient is under the influence of the mercury, or if the case is not very severe, you can give Liquor Hydrarg. Perchlor. with Iodide of Potassium, and I generally combine it with the Tinct. Cinchonæ Co. Sometimes you will find it useful to give the iodide in copious draughts of barley water; under such circumstances patients will sometimes tolerate it when they would not otherwise do so. Iodide is *the* drug to use in these cases, and we can understand how it acts. You can see a gumma which is superficial disappear under iodide, and there is no doubt the same thing takes place when the gumma is situated on the membranes of the brain, or in its substance.

I would therefore point out to you the importance of recognising the early symptoms of cerebral syphilis, and ask you to remember that cerebral syphilis can come on within even a few months of infection. In fact, it has been stated on good authority that cerebral syphilis has occurred while the primary sore was yet unhealed. There are many cases on record where it has occurred within three months of infection. Beaudoin collected in Fournier's clinic twenty-six cases in which the symptoms came on within three to six months of infection. Though I have seen a good many cases of cerebral syphilis, you must not suppose that this is a common result of syphilis. As a matter of fact, cerebral troubles only occur in about 2 per cent. of syphilitic cases, and about 2 per cent. suffer with parasyphilitic affections, either tabes or general paralysis. These diseases are considered to be common, partly because tabetic cases go about from hospital to hospital for fifteen to twenty years even, and as they are seen over and over again, they are apt to be looked upon as fresh cases, and to give an undue impression of their frequency. I have been round to many of the infirmaries of the metropolis lately, and I have not found nearly so many cases as I expected.

WITH SIR WILLIAM BENNETT IN THE WARDS OF ST. GEORGE'S HOSPITAL.

A PERFECT RESULT FOLLOWING UPON EXCISION OF THE KNEE.

HERE is a young man *æt.* 16, whose left knee-joint has been excised. You will remember that last week in Drummond Ward, when speaking of a case of tuberculous disease of the knee, in which I proposed excision, I said that excision at a very early period of life is not generally considered to be a good operation, not because it is difficult to bring about an immediately good result, but because the tendency towards subsequent bending of the limb at the point of operation which exists in these young subjects results sometimes in rendering necessary a second or even a third operation for the rectification of the deformity.

I have asked this patient to come up to-day for your examination, as he shows a perfect result after excision of the knee, the operation having been performed fifteen months ago. No change of an unfavourable kind is now likely to occur, and you may consider his present condition to be final. He is sixteen years of age; the girl in the Drummond Ward, of whom I was speaking, being several years younger. The local conditions were precisely the same in the two cases. I consider the result of this case to be perfect, because the limb is absolutely straight, the union is perfect, the position of the foot is good, and the patient can walk almost without limp. There is an important point about the singularly good gait which follows in a really successful case of excision of the knee. The shortening which arises from the removal of the articular surfaces of the knee-joint, provided that it is not excessive from interference with the epiphysal cartilages, is not a disadvantage to the patient in walking; on the contrary, it is a great advantage, because the leg can swing backwards and forwards without catching the ground. If you notice a man who has a stiff knee from ordinary ankylosis without shortening, you will see that in order to walk he has to throw his leg out in a very awkward way, because it is too long for the antero-posterior swing, so that it is always liable to catch in the ground, or in the

dressings of persons who are passing, unless a boot with a thick sole be worn *on the sound limb*.

This youth, you see, walks almost without a limp, and in a short time he will be able to walk any distance. Fifteen months have elapsed since his operation, and I should say that in spite of the excellent result which you now see there was no pegging, or wiring, or other artificial means adopted to keep the ends of the bone in apposition except ordinary splinting. Neither was there any plaster of Paris used. Pegging and wiring or screwing in these cases of excision of the knee is always unnecessary and sometimes harmful if the operation is skilfully done and if ordinary care is taken to manage the cases properly afterwards. If at the end of the operation the bones can be placed without difficulty in perfectly good position, by which I mean in contact over the whole of the cut surfaces, the limb being absolutely straight, or, better, a little over-extended, you should have no difficulty whatever in keeping the bone in position, and getting a result like this. It is only in cases in which the apposition is not quite good, or in which, for some reason or another, there is some peculiar defect, either about the patient, about the operation, or in the management of the case, that an immediately good result cannot be obtained without fixation by pegs, etc.

The question which arises in any case of this kind is whether the leg will remain straight permanently in the perfect condition in which you see it now. In this instance, as the patient is as much as sixteen years of age, the prospect is very favourable, but even now there is just a possibility that some bending may take place in spite of careful management in the course of the next twelve months. In younger patients (the younger the patient the greater is the difficulty) the tendency to bending at the point of operation is very strong, and cannot be obviated by the use of screws, pegs, or other contrivances of a like sort. It is not altogether uncommon to see a case of excision of the knee in a child (say six or seven years old), the condition of which at the end of six months or a little later has been perfect, return to the hospital a couple of years or more later on with a knee considerably bent. And it is sometimes, under such circumstances, said, "Here is an example of a case of excision of the knee which has been mismanaged, or in which some-

thing has happened which should not have been allowed to occur." You must at once please understand that although no doubt in a certain number of cases some fault in the patient or in the management of the case has to do with the defective results which we see at distant times, it does not always follow in cases of excision of the knee that there has been any mismanagement or any fault at all, either in the patient or on the part of the surgeon or practitioner. Now although this man is, as I have just stated, in a perfect condition now, if he were to come here again a year from now I could not guarantee that he will be in as perfect a condition then as he is now. It is possible, although most unlikely seeing his age, that at the end of another year the limb may be bent at a slight angle, in spite of the union being so sound and the whole thing being so satisfactory. What is the explanation of that? If this patient presents a less satisfactory condition at the end of the year from now, obviously it will not be our fault; we have conducted the operation and its after-management in such a rational way that at the end of fifteen months it is as perfect a case as one could find. You will bear in mind that in dealing with these cases of excision of the joints in young subjects we are particularly careful, especially in the case of the knee, to avoid taking away any portion of the epiphysal cartilages; we leave those as a matter of course, and it is to the irregular growth of the epiphysal lines that the deformity in these cases is apt to be due. Speaking generally, you will find that the growth of a bone at the epiphysal line is, in its rapidity, in direct proportion to the amount of freedom with which it is allowed to grow; in other words, if at any part of an epiphysal line the amount of pressure is in excess, there will be less rapid growth than in any part in which pressure is less or non-existent. In this case we, as a matter of course, did our best to save the whole of the epiphysal lines. The proper growth of the limb depends entirely upon the integrity of the epiphyses, and if we did our operation properly the epiphyses have been left intact. In the case of the knee there is a great tendency (the younger the subject the greater the tendency) for the growth of the epiphysis to be irregular for the following reason: the parts about the posterior aspect of the limb behind the knee-joint are very

much stronger and very much more intimately connected with the bones than those about the anterior aspect of the joint, the result of which is that when growth begins the pressure at the posterior aspect is very much greater than it is at the front, added to which is a tendency to contraction of the strong flexor tendons upon the leg, which, even if they are freely divided at the operation, soon re-unite. In the aggregate the result, therefore, is that the posterior part of the growing epiphysial line is subjected to greater pressure when the epiphysis begins to grow than the anterior part, where there is very little pressure at all in consequence of the different anatomical connections of the parts. The result of this is that the growth of the epiphysis takes a wedge-shaped form, with its base forwards, the leg being bent backwards (flexed) to a degree which is determined by the thickness of the (anterior) base of the wedge. The exact degree of the thickness of the base of the wedge depends upon the amount of tying down of the parts about the back of the bones. If the amount of pressure both in front and behind is equal, the epiphysial line will grow evenly, and no tendency to angular deformity will occur. On the other hand, if the parts behind are matted more tightly than in front, the growth of the epiphysis will be of a wedge shape, the anterior part growing more rapidly than the posterior. The important point which you have to realise is that in very young growing subjects there is, in spite of all that you may do, a tendency, which amounts to a certainty in those patients under eight years of age, to angular deformity after excision of the knee, from the causes which I have indicated, which are practically, in the vast majority of cases, beyond the control of the surgeon. The fact, however, that there is this tendency, which, it must be borne in mind, continues until growth is complete, affords no valid objection to excision as such, as the recurring deformity can be met by subsequent operations, the original alternative having been amputation. It is nevertheless a very important point in the matter of prognosis, and should always be pointed out to parents in cases of the kind in very young subjects. After the period of growth has expired, and ossification has become complete, all tendency to deformity other than that which can be controlled by the surgeon ceases, so that the final

condition depends on what is done at the latest stage. The case before you is a most excellent example to see, because you do not get the opportunity of seeing these cases every day, as it is so difficult to keep in touch with them after they leave the hospital.

SARCOMA OF TIBIA.

This unfortunate young man presents an interesting example of sarcoma of the tibia. You, of course, know that it is a serious condition. The man's age is twenty-four, and he is a gardener. He is of an age at which sarcoma is prone to develop. Until five months ago he was perfectly well, that is to say, nothing had called his attention to any abnormality up to that time. Then he noticed a little rounded swelling below the outer side of the knee, which was accompanied by some aching pain. He thought very little of it, but the swelling has gradually increased; latterly the pain has become a little more evident; it has now begun to make him limp, and he is less able to follow his employment than formerly. You will see that he is a healthy-looking fellow, well nourished, and not apparently one who is the subject of any wasting disease. Upon examination there is evident, occupying almost the upper half of the leg, a large nodulated tumour. It seems to be limited to the outer side of the leg; it is covered by a few large veins, but they are not a very prominent factor in the case. As I said just now, the tumour is distinctly lobulated, and if you feel along its margins you will notice that they are very abrupt, so that in parts the finger leaves the tumour suddenly at its edge. It is quite inseparable from the tibia itself, and terminates very abruptly at the upper margin of the bone. It ceases, in fact, at the upper end where the periosteum is more particularly attached to the bone. The joint is obviously healthy. There is no bulging into the joint or anything of that kind; the movements are free, and there is no aching or discomfort of any kind in the articulation. The swelling is hard to the touch, and in certain parts nodules are clearly felt. This tumour is, I believe, a sarcoma. It is either a periosteal sarcoma, which would be either of the spindle celled or mixed spindle- and round-celled variety, or it is endosteal, *i. e.* a myeloid sarcoma. The importance of determining of which kind it is is very great. The myeloid (myeloma,

as it is now called) or endosteal sarcoma is comparatively unimportant compared with the periosteal. This tumour is very hard; it is very irregular, and, if it is periosteal, it has passing through it, as is very common in these periosteal sarcomata, a little scaffolding of calcareous material, and perhaps bone. There is calcified matter in it supporting the tumour. The only thing which raises one's doubts a little about it being a periosteal sarcoma is the peculiarity of a prominent nodule at its inner and lower part. On feeling this nodule you find the finger sinks into a sort of depression in its centre, almost as if it were going into a crater. If that sensation be accurate it shows that it is not a periosteal sarcoma, but that it is endosteal, and that it has grown up from the interior of the bone through that crater-like orifice, and is overlapping the bone and surrounding it, as these endosteal tumours will sometimes do when they get outside the bone itself. It is most important that this point should be decided as far as possible before any operation is attempted. The evidence against the growth being endosteal is the remarkable way in which the tumour is limited to the outer two thirds of the circumference of the bone. The inner side of the tibia is normal, and cannot be said to be expanded; in a case of endosteal growth there would almost certainly be some general expansion of the bone, and, of course, according to the books, if it were endosteal, you should be able to feel "egg-shell crackling." But although the books quote this as being a pathognomonic sign, I have examined many of these cases, but in very few of them have I felt egg-shell crackling. The absence, therefore, of such crackling does not go in the least to show that the growth is not endosteal, although if it were present it would be a strong affirmative indication. You will notice in this case how very abruptly the tumour terminates, *not at the extreme upper edge of the tibia, but at the point where the periosteum becomes intimately attached to the bone, just below its upper edge.* I suppose you know that especially in the long bones, as you approach the articular surfaces, the periosteum becomes so intimately attached that it is practically impossible to separate it, a state of affairs quite different from that which holds good about the central portions of the shaft. The growth in periosteal sarcoma is generally abruptly defined along the line of this intimate periosteal

attachment; on the other hand, endosteal growths near the bone ends of the size arrived at in this case would certainly be associated with expansion of the bone to its extreme limit, a bulging into the joint resulting. There is nothing of that kind here, the tumour terminates abruptly before reaching the extremely upper end of the tibia. That points to it being a periosteal sarcoma. The most important question, of course, is with regard to the treatment and prognosis of the case. The diagnosis is not a certain one, but I shall be surprised if, in face of the signs to the contrary, the growth after all comes from the inside of the bone. This crater-like depression I have called your attention to is suggestive, but, at the same time, is probably deceptive.

The question of which kind of sarcoma it is can practically be settled by the X rays, so we shall have a Röntgen picture made before deciding on our course of procedure. If it is periosteal sarcoma the prognosis is bad, if endosteal growth the prognosis is good. The treatment will differ a good deal in the two conditions. Supposing the growth to be endosteal, I am afraid it has gone too far for enucleation, and the alternative would be to resect the upper half of the tibia, or to amputate through the knee. Now resection of the upper half of the tibia in a case like this would leave such a faulty limb that it would probably be as well to amputate through the knee. So that if it is found to be endosteal, I think amputation through the knee-joint will be the course to pursue, as the necessary flaps could be safely fashioned from the parts overlying the tumour; if, however, the tumour be a periosteal sarcoma, the making of flaps from parts overlying the growth would be out of the question, and amputation must be performed through the thigh, preferably through its middle, or higher. It is a sound practice in removing a limb for periosteal sarcoma to leave, when possible, a joint between the disease and the seat of amputation.* Beyond these points the case presents nothing of special importance. I must, however, remind you that it is extremely bad practice in cases of carcinoma and sarcoma, either before the operation or at the time, to lay

* The X rays confirmed the diagnosis of periosteal sarcoma, and amputation was performed through the middle of the thigh. Pathologically the growth was a spindle-celled sarcoma.

open the growth itself for diagnostic purposes, because in doing so you will lay open lymphoid tissue and lymph channels which may readily absorb the sarcoma or carcinoma. Although it is with some surgeons a common practice to incise tumours for diagnostic purposes, it is a dangerous habit, and you should endeavour to arrive at the right line of treatment without opening the growth. Do not lightly incise these tumours for purposes of examination for fear of further infection.

RECURRENT FRACTURE OF THE PATELLA.

Here is a man who has twice had fracture of the patella. He was in hospital for the first time in 1896, under the care of another surgeon, and the bone was wired immediately. He left the hospital with the knee comparatively stiff, as he could not stand the *necessary manipulation*. The case had done very well, the wire had caused no irritation, but there was very little mobility in the knee. Shortly after he left the hospital he fell down and bent the knee violently, and from that time up to a few weeks ago he was well and had free movement in his knee. He had accomplished the popular operation of bone-setting for himself. On October 7th (a month ago) he was knocked down by a runaway cab, the fall being not so fortunate as the former one, as it caused him to break his knee-cap for the second time. He came at once into the hospital with the two fragments of the bone lying a considerable distance apart, the wire which was used for the former operation being easily felt broken and curling up under the skin. There was a great deal of effusion for a time, and I waited, as I generally do, for a week before attempting the re-wiring of the fragments. I then cut down upon the patella, and after removing the old wire—which was rather a thin one—brought the fragments of the bone together again with a stout wire. The second fracture had taken place at the site of the old one, and upon examining the ends of the fragments they were found to be quite free, the cancellous tissue being thoroughly opened up. There was no necessity, therefore, to do anything like a formal freshening of the bone ends. The fragments were fortunately about equal in size, which is an excellent state of affairs for wiring. In the former operation a vertical median incision had been employed; in my operation I turned up, as I am in the habit of doing, a flap from the outer

side, which I think is far preferable, as it leaves the wire remote from the wound. The case has done very well; nothing material has happened; the wound has now healed; there has been a little oozing from it from time to time, but in spite of that slight defect you will notice that we began to use passive movement very early, a fact to which I particularly wish to call your attention, as there is no reason, if ordinary discretion is used, to postpone passive movement until the wound is sound. These wounds sometimes heal slowly, and if movement is delayed until they are absolutely sound valuable time is often lost, and unnecessary delay follows in obtaining free movement in many cases, and in some free movement may be thus entirely prevented. This operation was done three weeks ago, and you will see that the movements of the joint are perfectly free up to a right angle, and the range of movement is increasing daily—an excellent result considering the unfavourable nature of the case. A most important detail in obtaining thoroughly good movement as quickly as possible is early passive movement of the patella, to avoid any matting around its margins. Should any adhesion of the patella occur, a great obstacle to perfect and speedy natural movement arises. This movement of the patella cannot be commenced too soon after the operation. This detail is of equal moment in cases treated without operation, and I am still of opinion that a considerable percentage of cases can be perfectly well treated without operation. There is, for example, an old woman in the Wellington ward whom we are treating without operation, and some of you will remember how particular I was to insist upon the fragments, whilst pressed as closely together as possible, being freely moved laterally and vertically every day. It is now about a fortnight since the patient's admission, and free passive movement at the knee is now possible without any separation of the fragments. So long as the patella is thus kept moveable, as it is in the patient I have mentioned, you can go on with passive movement almost to any extent without fear of doing harm, because if properly held during the bending of the knee, as I show you now, the upper part of the patella, when the leg bends, goes with it, and slides down on the intercondyloid notch of the femur in the ordinary way, no separation of the fragments resulting. Supposing, however, that the patella had been allowed to become in any way fixed by adhesion

the joint could not have been bent without a wide separation of the fragments occurring. At later periods, again, there is danger of refracture occurring if the patella becomes fixed, when attempts at bending the knee are forcibly made. Returning to the consideration of the case before us you will note that the fragments are of equal size, an ideal arrangement for the radical operation by wiring, in which the more nearly the fragments are equal in size the better would be the prospect of the operation. Sometimes you may have to deal with cases in which one fragment is very large and the other one very small, indeed, so small as to be almost non-existent. I have had to deal with cases in which the upper fragment has been nothing more than a little nodule of bone. Now, in cases of that sort, what is the best way of applying the wire? In cases such as the one before us there is, of course, no difficulty at all. The patella is drilled through the substance of both fragments, the wire being then outside of the joint cavity altogether. It is obvious, however, when one fragment is very small that to drill it in this way would be useless, as it is so small that the wire would break away from the little piece of bone itself. What then is the best practice? There is, I think, no doubt that the best thing then is to pass the wire through the quadriceps tendon above, carrying it under both fragments, and then through the ligamentæ patellæ below, bringing the ends together over the front of the patella in the usual way. I think one wire is enough for the wiring of any broken patella, especially if the wire be of stout size; two wires, as used by some operators, make the operation a little longer, and offer no advantage so far as I know; indeed, in a case in which I used two wires very carefully acute necrosis, apparently aseptic, of the patella followed, the only case I have ever seen. I thought that it was probably due to the nutrition of the bone having been too much interfered with by the double drilling, and that the vitality of the bone was therefore not sufficient to maintain its integrity. Of course, however, the occurrence may have been a mere coincidence. I have never found it necessary to use two wires to effect a thoroughly efficient approximation of the fragments.

Please do not imagine that perfect results are not possible without wiring in fracture of the patella. We know that practically as good results

as those obtainable by wiring can in some cases be obtained without, but as a rule it is said that there is no perfection in them, because there is fibrous union, but for practical purposes very strong fibrous union seems to be as good as bone. It is said by the very strong advocates of the wiring treatment of fracture of the patella in all cases, that patients who are treated without operation are very much more liable to fracture of the opposite patella than patients who are treated by the wiring operation. I have, of course, seen a certain number of patients myself who have had fracture of the opposite patella when the originally fractured patella was treated on the old lines. But our experience of the wiring treatment is not so large as our experience of the non-operative treatment. I have seen a patient suffer a fracture of the opposite patella after a fracture treated by wiring, and I should say that recurrent fracture of the same patella is relatively more frequent than the rupture of the fibrous union resulting from the non-operative method. The liability to a second fracture in cases treated by operation depends, apparently, upon one of two conditions: first of all, defective union between the fragments, which is more frequent than is commonly supposed, and secondly, the use of wire which is too thin. Of course if the bony union were thoroughly sound and hard, naturally no permanent wire at all would be required to help the completion of the case. If, on the other hand, union is not so very sound, as may happen, for example, if the fragments are very unequal in size, you can see that although the union between the fragments would not be perhaps sufficiently strong in itself to keep the bone intact under great stress, still, if a sufficiently strong wire were supporting the parts, they might remain intact, although the union itself may be comparatively incomplete or weak. If, on the other hand, the wire were insufficiently strong to bear the secondary strain the patella might give way again. Finally, it is almost unnecessary, I presume, to remind you that it is always better when you can, to leave the wire *in situ* permanently and not to remove it, because the wire, if it causes no harmful irritation, is backing up a bone which might be weak. If, on the other hand, the wire is removed as a matter of course, if the union be weak it loses the support of the wire, and misfortune may come in a case which would otherwise go well.

THE TREATMENT OF HEART DISEASE.*

By G. W. EUSTACE, M.A., M.D.

TO-DAY I wish to lay before you the consideration of a very common, but no less interesting on that account, class of diseases, namely, those of the heart, and to confine my remarks to a few points in connection with their treatment.

Diseases of the heart are no exception to the rule that the more accurate the diagnosis the more successful will be the treatment; to ensure this accuracy, diagnosis must not only be particular, it must also be comprehensive, the secondary results being often of greater importance than the primary lesion; this is a self-evident proposition, and yet failure in treatment is most frequently due to its non-observance.

I do not propose to deal further with the subject of diagnosis beyond stating that the presence or absence of bruits is often of far less importance in considering treatment than other equally evident signs of disease.

I merely allude to this because it seems to me that too much attention is frequently bestowed on the presence of murmurs to the exclusion of the condition of the chambers of the heart, the state of the cardiac muscle, and the degree of blood-pressure in the large arteries and veins.

From a therapeutic point of view, diseases of the heart may be divided into two stages:

Firstly, the stage of inflammation, either of the heart or pericardium, or of some organ or tissue, inflammation of which reacts on the heart through the circulation; and secondly, the stage of mechanical failure of the heart. That both stages are often present at the same time does not lessen the value of this distinction.

Taking first the stage of inflammation. Treatment will depend in a great measure on the cause of the inflammation and on the part affected, but the object of treatment will in all cases be the same, to bring about resolution of the inflammatory process as speedily as possible, and thus avoid, or at least postpone, the stage of mechanical failure.

The indications for treatment during the first or

inflammatory stage will be furnished by keeping in view three points: (1) the site of the inflammation, (2) its cause, and (3) the natural result if untreated.

In this light let us first consider the treatment of pericarditis. Here the inflammation is not confined to the pericardium, but affects the underlying muscle fibres; the contractile power of the heart is thus diminished, hence the work which the heart has to do must, by treatment, be correspondingly diminished; this will be effected by complete rest, by giving a mercurial purgative, followed by the administration of the vaso dilators to bring about a general lowering of the blood-pressure, by the avoidance of certain meat extracts, whose only effect is to increase the arterial tension at the expense of very uncertain nutritive value, and by the greatest caution in the use of the digitalis class of drugs; at the same time the heart's action must be sustained by giving light, easily assimilated food, especially milk, and by the administration of strychnine and ammonia if symptoms of cardiac failure threaten.

When pericarditis is associated with acute rheumatism, it is right to continue, or even to increase, the dose of sodium salicylate, it being assumed that in this case both are due to the same specific cause; but in pericarditis, arising independently or in the course of other diseases, little, if anything, can be done in the way of direct treatment.

The natural results of pericarditis are the formation of fluid, serous or purulent, in the pericardial sac, and pericardial adhesions, either between the two layers of the pericardium or between the parietal pericardium and the chest-wall.

If the amount of fluid is excessive or persistent, iodide of potash should be given and iodine painted over the precordium; blisters may be tried, but their effect is uncertain and seems sometimes to increase the fluid present; only if this is so excessive as to threaten a fatal issue or if it is purulent should paracentesis be performed.

With regard to adhesions, once they have formed it is doubtful if anything can be done to absorb them; something may, however, be done to stretch them, as soon as it is clear that the cardiac muscle is regaining its vigour, by the judicious administration of digitalis, and by graduated exercises of the Scholt type, to increase the force of the heart-beat; but here great caution must be

* Delivered before the West Sussex District of the South-Eastern Branch of the British Medical Association.

observed lest too much be attempted and dilatation result.

The only special symptom in pericarditis requiring treatment is pain; this is best met by the application of from four to six leeches over the precordium or an ice-bag, or by the hypodermic injection of morphine and atropine.

Taking next the subject of acute endocarditis, treatment will be directed to preventing permanent damage to the inflamed valves by in all cases insisting on complete and often prolonged rest, and in endocarditis of rheumatic origin, and possibly in endocarditis arising in the course of chorea or scarlatina, by giving sodium salicylate, and during convalescence, iodide of potash, arsenic, and iron.

Malignant endocarditis is an incident in a general septic infection with a very fatal tendency, and treatment directed to the condition of the heart will be that of the general disease; stimulants will be early required to avert cardiac failure and to assist the struggle against the generalised poison.

In endocarditis when the valves show signs of having sustained permanent damage, efforts should be made to assist compensatory changes in the heart, the most important of which is hypertrophy, and in this connection it may be stated that hypertrophy is probably never a primary condition or a disease at any time; its presence is an indication of cardiac vigour and a necessary part of compensation, and in cases of hypertrophy where inconvenience and distress are caused by the violent contractions of an hypertrophied heart, relief must be sought by diminishing the arterial tension with the vaso dilators and not by the administration of such drugs as aconite, given with the object of directly weakening the too forcible action.

To assist compensation complete rest for several weeks, and in the case of aortic regurgitation, even for months, will be required; the quality of the blood must be improved with iron and arsenic, by giving mercurial purgatives to eliminate waste and injurious products, by limitation of fluid and careful attention to diet, and if the arterial tension is unduly high by giving the spirit of nitrous ether and the acetate or citrate of potash or nitroglycerine. During convalescence, and until compensation is fully established, exertion must be strictly limited, lest marked dilatation occur; I use

the word marked advisedly, because it is probable that in permanent valvular disease a slight degree of dilatation always accompanies the hypertrophy, and is beneficial, and not an evidence of weakness.

When compensation is fully established, treatment will be directed to avoiding recurrence of inflammation and to maintaining compensation; on these two points the patient's future will depend, the situation and nature of the lesion determining the exact preventative treatment required.

If the heart trouble has arisen in connection with acute rheumatism the greatest care must be taken to avoid other attacks, and slight pains in the joints must receive far greater attention than would otherwise be given to them.

If the mitral valves are the seat of the damage the care of the right ventricle will be the chief essential of treatment. To this end, the relief of hepatic engorgement, graduated exercise in the open air, especially at high altitudes, by causing increased action of the heart with diminished pulmonary resistance will contribute; conversely, anything which increases the strain on the ventricle, such as an attack of bronchitis, will act detrimentally, and probably lead to dilatation, and unless this and the special indications for its use, to which I shall later refer, are present, the digitalis class of drugs is best avoided.

If the aortic valves are the seat of the damage, whether resulting in narrowing or in incompetence or in both, exercise must be strictly limited, digitalis as long as mitral regurgitation is absent avoided, and treatment chiefly directed to keeping down peripheral resistance and maintaining the general health. If both mitral and aortic disease co-exist our efforts will consist in a combination of the treatments suitable for both, attention to the right side of the heart predominating.

Chronic endocarditis, occurring usually after middle life and due to atheromatous change in the valves and tendinous chords, the result of syphilis, or of continued high arterial tension from gout, chronic renal disease, or excessive muscular effort, will be met by treating these affections as the primary causes of the cardiac condition, iodide of potash being specially indicated where there is a marked specific history.

Myocarditis is met chiefly in connection with peri- or endocarditis, or as suppurative inflammation the result of pyæmia; the form associated

with rheumatic pericarditis is the only one that can be looked on as curable, and here the treatment for the relief of the accompanying lesion will be applicable, the support of the failing heart being the main indication.

Fatty degeneration of the heart must be carefully distinguished from excessive fatty overgrowth of the heart, a part of general obesity. In true fatty degeneration the muscular fibrillæ themselves are the seat of minute fat granules, which replace the true sarcous elements, and rob the muscle of so much of its contractile tissue. When fatty degeneration arises in the course of other diseases such as typhus fever, diphtheria, or influenza, the degeneration will usually cease with the subsidence of the primary disease inducing it; treatment will consist in supporting the weakly acting heart by stimulants such as alcohol, ammonia, or strychnine, and in preventing whatever requires sudden cardiac effort; in convalescence from acute disease this should always be borne in mind, lest sudden death occur, or dilatation be provoked; and permission to return to ordinary habits of life should be withheld until all traces of cardiac weakness have disappeared. In fatty degeneration, the result of atheroma of the coronary arteries, or of previous myocarditis, the disease must be looked on as progressive, and careful rules as to exercise and food followed, by the avoidance of constipation, of large and hurried meals, especially before retiring to rest, of undue exertion, of making efforts with the breath held, and of mental excitement. Cardiac stimulants will always be required, especially ammonia and strychnine; digitalis must be given with caution, and only in cases where the heart-beat is frequent and irregular, with evidence of dilatation.

Persistent high arterial tension must claim our attention for a few moments. When such a condition is present even although there may be no signs of renal or pulmonary disease, means must at once be adopted to lower the abnormal blood-pressure, because sooner or later the heart will yield to the increased strain thrown upon it, and valvular incompetence or cardiac dilatation result. Increased systemic resistance is usually associated with gout or chronic renal inflammation, and here it should be remembered that the condition of the heart in chronic renal disease determines which form of kidney will result, the large white kidney

being associated with early failure of the heart, and the small granular kidney existing with cardiac vigour and hypertrophy.

The treatment of persistent high tension will consist in the limitation of nitrogenous food, the suspension of alcohol, owing to its general tendency to interfere with due metabolism and elimination, and to bring about degeneration of structure; careful attention to the action of the bowels, so that waste and injurious products may be removed from the system, whose circulation in the blood is a constant source of high tension; indirectly, by improving the quality of the blood with iodide of potash, iron, and arsenic, and directly, by the administration of such drugs as Spirit. Ætheris Nitrosi and nitro-glycerine. When strain is thrown on the heart by increased pulmonary resistance through chronic bronchitis or disease of the lungs, treatment will be directed to the relief of the primary disease causing it.

This concludes a brief survey of the indications for treatment in the first or inflammatory stage of heart disease.

(To be concluded.)

Spasmodic Torticollis.—After noticing first the symptoms, clinical history, ætiology, pathology, prognosis, and treatment, Hamann ('Buffalo Med. Journ.,' December) gives his own operative experience with neurectomy, excising the posterior divisions of the three upper cervical nerves, also cutting the muscles. He insists on the ætiologic importance of frequently repeated muscular contractions in the production of disease, the uselessness of non-surgical measures, at least in advanced cases, the safety of the operation, the absence of impairment of motion after it, and the good chance of cure or improvement. He urges the performance of radical and extensive operations—excision of the nerves and divisions of the muscles.—*Journ. Amer. M. A.*, December 28th.

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A CLINICAL LECTURE

ON

CASES ILLUSTRATING VARIETIES OF EPITYPHLITIS.

Delivered at University College Hospital,
December 20th, 1901.

By ARTHUR E. BARKER, F.R.C.S.,

Professor of Surgery at University College, and Surgeon
to University College Hospital.

GENTLEMEN,—The four cases of epityphlitis now before you, on which I have operated within the last few weeks, offer an excellent opportunity for a few remarks on some of the varieties of the disease and its surgical treatment, of which I propose to avail myself to-day. Each is an example of a different anatomical position of the epityphlon or vermiform appendix, and each of a different form or degree of inflammation.

Let me point out to you first the three or four common anatomical situations of the organ we are concerned with, all illustrated by the cases convalescing before you.

The first, and, as I believe after operating on many scores of such cases, the commonest, is that which you saw when the abdomen was opened in the little boy now in Bed 9. Here the appendix (Fig. 1, 1), starting from the point where the two tænia meet on the fundus of the cæcum, just below and internal to the anterior iliac spine, runs mostly upwards and inwards towards the umbilicus, lying more or less upon the small intestines, and in its inflamed state, with the pus around it, forming a comparatively superficial swelling.

A second was seen in the case now in the private ward getting well. Here the epityphlon pointed horizontally towards the opposite groin (Fig. 1, 2), lying almost immediately beneath the abdominal wall and upon the intestines, so that the inflammation and pus around it were also superficial.

In the third case, operated on ten days ago, and now in Bed 1, a long appendix ran downwards and inwards over the brim of the pelvis, and lay behind the bladder covered by small intestines

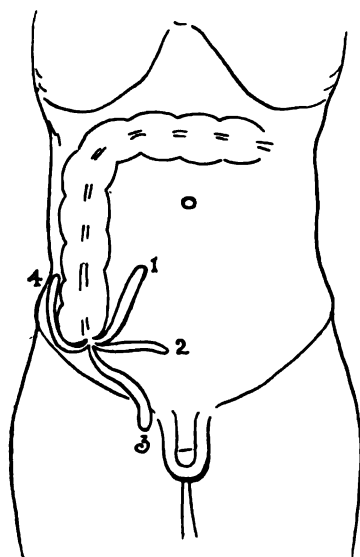


FIG. 1.—1, Commonest position; 2, next in frequency; 3, next in frequency; 4, least common.

(Fig. 1, 3). This was a very bad case, and came to us with general peritonitis in the lower half of the abdomen and flanks. The whole of the true pelvis was full of stinking fluid, so that no particular point of induration could be made out from the rectum, while anteriorly a clear note and no induration could be made out through the parietes.

In the fourth case the epityphlon lay buried behind the cæcum, running upwards towards the right kidney (Fig. 1, 4). Here there was exquisite tenderness inside the anterior superior iliac spine, but there was no tumour and a clear percussion note was obtained from the overlying cæcum. These cases, it happens, occurred in the order of their frequency.

To reach the appendix or the abscess around it in these several cases it may obviously be necessary to make a different incision for each. In the first and second cases it would be proper to aim at opening the abdomen through the external and internal oblique muscles by separating their fibres just outside the border of the rectus, or through the sheath of the latter (Fig. 2, *a*). In the third case, which was rather obscure, and when the whole of the lower part of the abdomen was full of foul fluid, I thought it better to make the incision

in the middle line (Fig 2, *b*) so as to be able to reach both sides and command the cleaning out of the pelvis; and from this incision the appendix, as it dipped behind the bladder, could be easily reached and removed. These pelvic varieties are, as a rule, obscure, and I recall a recent private case, supposed to be an ovarian cyst with a twisted pedicle, when, on making the median incision, I found a very long appendix reaching over to the *left* side of the bladder. It was not difficult to remove, and the patient recovered rapidly.

In the fourth case, where the appendix lay behind and external to the cæcum, the incision should be placed just internal to and running (Fig. 2, *c*) above the anterior superior spine, or the pararectal incision carried a little high may be done. In this case I made it a little lower than was right, and had some difficulty in extracting the organ without tearing it. But even here it was remarkable what one could do by pulling the external lip of the wound outwards with a strong double hook, and I got out the whole organ intact, although it was greatly diseased.

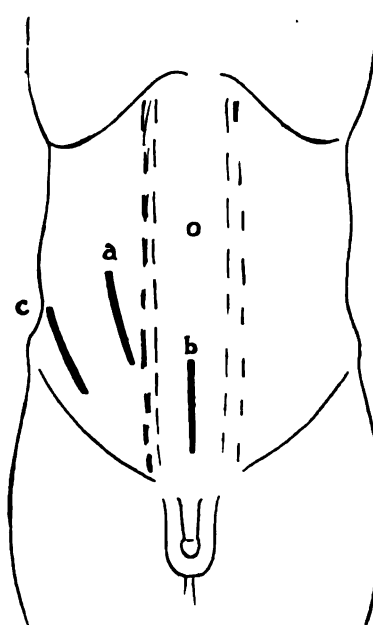


FIG. 2.—*a*, Common incision; *b*, median incision; *c*, retrocaecal incision.

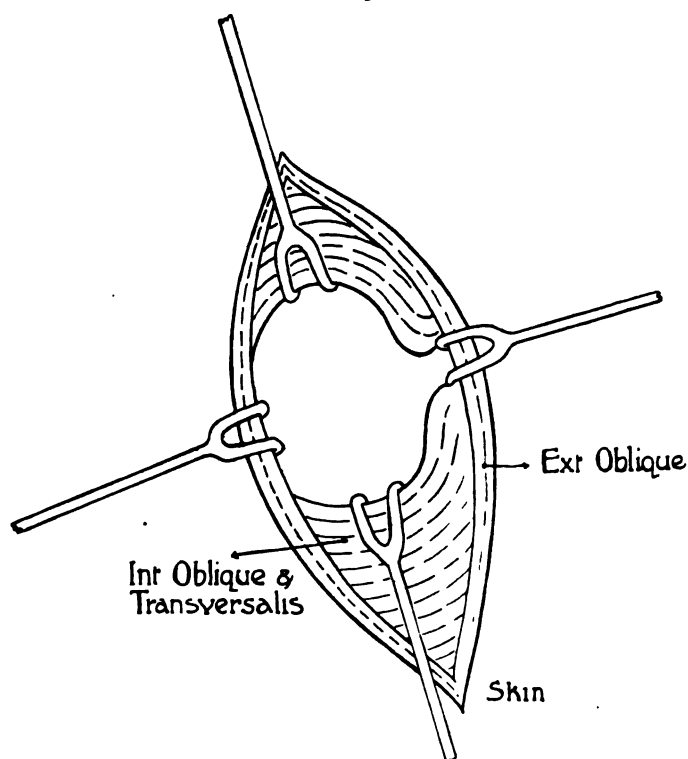
And now let us see what were the pathological forms in these four cases. What was actually found *in vivo*?

In the first case, in Bed 9, the boy on admission was found to have a swelling about the size of a lemon lying in its long axis, parallel to Poupart's ligament, and having one third above the line uniting the iliac spine and umbilicus. This swelling was comparatively superficial and fairly well defined laterally. It was exquisitely tender, and over it there was diminished resonance to percussion, the rest of the abdomen being normal in this respect. Nothing abnormal was felt by the rectum. An incision was made nearly parallel to

The appendix was easily stripped out and removed in the usual way. It was much diseased at its free end, and almost, if not actually, perforated. On being slit up its proximal portion was fairly sound, but the distal was ulcerated and rotten.

Here, then, was a case of ulcerative disease of the mucous membrane of the appendix *slowly* perforating after adhesions had formed around it. These adhesions were firm enough to prevent general infection of the abdomen, but not the formation of a foul abscess.

FIG. 3.



the outer border of the rectus, and about half an inch external to it; the fibres of the ext-oblique were separated by pulling with hooks outwards and inwards; the fibres of the int-oblique and transversalis by pulling upwards and downwards without dividing them (Fig. 3), and the peritoneum was opened. Immediately thick foul pus escaped to the extent of about a couple of ounces. On passing in the finger the appendix was felt running towards the umbilicus at the bottom of the abscess. The latter was limited all round by firm adhesions.

In the second case, in the private ward, the onset of the disease had taken place five days before with colicky pains in the caecal region and rise of temperature. When I saw the patient on the fourth day I found a large swelling in the iliac fossa, quite superficial, dull to percussion, and very tender. On making an incision half an inch outside the margin of the rectus in the umbilico-spinous line, and separating the muscular fibres, I came at once upon pus of the foulest kind. When this was washed out with normal saline solution the

appendix was seen lying at the bottom of the abscess and pointing towards the opposite groin. It was quite gangrenous and converted, except for the last inch of its tip, into a grey slough. It was tied at its base and removed. The free end was purple and discoloured, and showed a couple of perforations, from which some stiff bristles protruded, which looked as if derived from a toothbrush. The abscess around was well defined, and was not further interfered with.

Here the perforation appears to have been *slow* and to have produced a more or less plastic peritonitis before the actual rupture. Within the included area pus was formed when the perforation occurred, and the appendix, bathed in this very irritating matter, sloughed as a whole. I ought to add that just before the drain opening closed a day or two ago, a concretion, the size of a small date stone was discharged from it. This looked like calcium phosphate encrusted round some hairs or bristles which protruded from one end.

In the next (third) case, which was the worst, the patient came to hospital after a four days' illness with general peritonitis. His abdomen was distended, and was all over tympanitic; nothing distinctive could be felt by the rectum. He had been very sick, and the doctor who sent him in stated that the vomited matter had at the last been stercoraceous. There was no special tenderness at any spot or any guide as to the starting-point of the peritonitis.

I opened the abdomen in the middle line and came at once on much foul fluid and lymph among the intestines which showed all the signs of acute peritonitis, being red, distended, and in part covered with large flakes of lymph. Searching at once with the fingers towards the right side of the pelvis, I came easily upon the appendix, which hung down behind the bladder under the small intestines. It was easily drawn out into the wound, being very long, and was separated at its base in the usual way. Its free end was sloughy and perforated. The pelvis contained quite half a pint of thin, foul fluid, turbid, and with flakes of lymph in it. This was mopped out, and still more and more flowed down from among the intestines. The latter were dried and cleared of lymph with gauze mops thrust among the folds. Some of the fluid was found in the left flank, less in the right, but the bulk of it was in the pelvis. Just

before putting in the stitches in the abdominal wall the few visible coils of intestine were washed over with normal saline solution, which was then at once mopped off with aseptic gauze. Here we had an aplastic process from the first started by a sudden perforation of the appendix which ran down into the pelvis below the small intestines and only mechanically limited by the clinging together of the latter. The position of the collection of fluid in the pelvis behind the intestines prevented its being discovered by percussion, the note being quite resonant in every part of the abdomen. The median incision enabled this fluid to be reached as well as the pelvic appendix, and also the fluid which reached as far as the umbilico-spinous line on each side at least.

Now in each of these cases we had not only a different anatomical position of the inflammatory focus, but also a different degree of inflammation if not actually a different specific poison.

In the first we saw a milder form of ulcerative epityphlitis leading first to adhesions round the appendix as the ulcers approached the serous covering of the organ, and eventually to an abscess with well-defined limits. In the second we had also an ulcerative process from the first, probably set up, or at any rate associated with, the presence of bristles. But this, although it was plastic at the start and was limited by adhesions, became gangrenous later on and destroyed almost the whole appendix, converting it into one long grey slough; and had the abscess in this stage been neglected and ruptured we should probably have seen the same general peritonitis as in the third case. In the latter the inflammation from the first was aplastic, probably owing to the virulence of the poison and sudden perforation. There was consequently no attempt at limitation of the process by adhesions, and general peritonitis was the result.

In the fourth case there was a violent non-ulcerative catarrh of the mucous membrane of the appendix without perforation. This had led to exudation of serum round the organ, which was soaked up with mops during the operation, but there was no limiting lymph. The appendix when slit up showed a dark purple mucous membrane, but no ulcers. It was also much thickened in all its coats by cedema and exudation.

It is clear, then, that there are several forms of the pathological process which I might classify for you under the following terms:

1. Simple catarrhal epityphlitis, subacute.
2. Acute septic epityphlitis.
3. Ulcerative epityphlitis with plastic adhesion.
4. Gangrenous epityphlitis, aplastic.

The first in this list is perhaps the most common, and leads to that clinical condition often called "appendicular colic," in which we find colicky pains over the cæcum with slight tenderness, but no definite tumour. Here there is found, when for any reason an operation is required (which is rare), a simple swelling of the lining of the organ and slight thickening of its coats, with a collection of mucus or muco-pus in its lumen, which is prevented from escaping into the cæcum by the swelling of the mucous membrane at the proximal end. Sometimes the amount is considerable, distending the organ into a bag of muco-pus, which may be on the eve of bursting when we see it. And doubtless such bursting is serious if the catarrhal products are very septic. In one of my cases recently I came upon such a distended empyema of the appendix, and just as I was getting it out the sac burst. Fortunately the wound and intestines were covered with gauze and the only ill-effect was some suppuration at one or two of the stitches where they were infected.

But, as a rule, such cases of simple catarrh of the epityphlon, if seen at the outset, can be treated by rest, fomentations, and mild laxatives. Under this the products of the inflammation are discharged into the cæcum and recovery takes place; but they are liable to recurrence, and ought certainly in many cases to submit to removal of the organ before some of the more severe forms make recovery problematical and render the chances of a sound scar remote.

And now a few words as to the ways in which these cases were operated on. The first two were, as you see, dealt with through a pararectal incision (Fig. 1, 1). This reached the abscess in each case easily, as it was superficial; and as the appendix was easily found in the pus in each case it was removed without disturbing limiting adhesions. I am one of those who do not search widely for the appendix in such cases. If it is easily found I remove it, if not, I simply drain with gauze packing and leave its removal for a subsequent interim

operation. Both these cases were drained with gauze and are now convalescent.

In Case 3, where the median incision (Fig. 2, b) was made and much mopping had to be done, I stitched up the wound completely, and hoped thus to tide over the immediate ill-effects of the operation better than with an open wound. I felt that if drainage were done in this case from the first that it would add to the risks without many corresponding advantages, for remember that in such a case you can only drain one corner of the peritoneal cavity with one drain, while the rest remains undrained. And now you see that although the stitches have inflamed and the two lower ones have given way the patient on this, the tenth day, is doing fairly well. His distension has subsided and his sickness is over, and though the temperature is high his pulse is only about 100°, and he can take food, while quite a large quantity of fæces is brought away by enemata, and none of his food is found when the stomach is washed out. He is, in fact, fighting the general peritonitis well, and seems in a fair way to conquer should no further complications arise.* In Case 4, though there was turbid fluid exuded round the inflamed organ, I did not think it desirable to drain, and, as you see, the wound now, on the ninth day, has healed by first intention. As to which case should be drained and which not it is hard to lay down positive rules. Experience must guide you, and each case in my own numerous series is treated in this respect on its own merits. Drainage is absolutely necessary in some cases, but I am sure it is not so in others. In many instances where there has been much intraperitoneal inflammation after cleansing the infected area I have stitched up the abdomen with the happiest results. In some it is true that subsequently one or two stitches have cut out and the wound has partially gaped, as in Case 3, but this has been no worse than had the drainage been provided for from the start, while I believe the immediate effect of the operation has been better. This certainly has been the case with Case 4 now, on the ninth day, practically well.

* This patient subsequently succumbed on the 18th day to septic pleuro-pneumonia on the left side. A great part of the peritoneum had recovered from its inflammation, but there was still a collection of pus on the right side of the pelvis which gave no signs of its presence during life. The three others are quite well.

There are many other details as to the after-treatment of these cases which are of interest, but I must leave them for subsequent consideration. One only I will mention in conclusion. In Case 3, where general peritonitis was present and the patient was vomiting frequently, I ordered that, morning and evening, he should be transfused hypodermically with half a litre of normal saline solution in addition to rectal enemata. The object of this was to supply the man's system with water sufficient to enable the kidneys to carry off the toxins which, presumably, he was absorbing from the peritoneum and excreting with the urine. There is reason to suppose that the kidneys are the great eliminators of these poisons. But where a patient is losing much moisture by the skin and lungs, and vomiting all that is given by the mouth, the kidneys have very little to work upon. A good deal can be absorbed by the rectum, but that which is passed directly into the circulation hypodermically must pass out mostly by the kidneys, and so tend to carry out the poison. Whether this be usual or not, the effect here has been most marked, and besides the pulse has been kept full. It has been all through quite unlike the small pulse of peritonitis. He has had these transfusions twice a day from the moment of operation until now, ten days later. The normal saline solution has also had 5 per cent. of glucose added to it, a means of supplying carbo-hydrates to the system which I have been using for more than a year now in such cases, I think with advantage.

Wound Diphtheria.—Tavel ('Deutsche Zeit. für Chirurgie') reports three cases; in a whitlow, an abscess of the muscles of the back, and paronychia of the index finger, diphtheria bacilli were found. The case histories are given in full. In none of them was the course of the affection at all different from what it would have been without diphtheria bacilli. There was absolutely no suspicion of a membrane. The diagnosis is impossible without a bacteriological examination, since most cases with the formation of a membrane are due to staphylococci and streptococci. Tavel concludes that the same kinds of disease may be caused by different bacteria, and the same bacteria may cause different forms of disease.—*Philadelphia Medical Journal*, December 28th.

A CLINICAL LECTURE ON SOME OF THE MEDICAL ASPECTS OF ADENOID VEGETATIONS AS EXHIBITED IN INFANCY AND CHILDHOOD.

Delivered at the Hospital for Sick Children, Great Ormond
Street, W.C., December 5th, 1901.

By ROBERT HUTCHISON, M.D., M.R.C.P.

LADIES AND GENTLEMEN,—I wish to direct your attention this afternoon to some medical aspects of adenoid vegetations as exhibited in the case of infants and young children. I shall assume that you are familiar with the local symptoms which are due to the presence of adenoid growths, and also with the chief surgical complications to which they may give rise, and shall direct my attention more to those diseases which accompany adenoids, and which, more strictly speaking, come within the cognisance of the physician. I do not propose to deal at all with the pathology of adenoids, because that is at present very ill-understood, and I can add nothing to what you already know on that aspect of the subject.

It will be convenient, I think, to divide the subject first of all into adenoids as occurring in infancy on the one hand, and in older children on the other. Perhaps it may be a surprise to some of you to hear that adenoids occur in infancy at all. Everyone knows that adenoids occur in older children. But I go further and say there is reason to believe that adenoids may be a congenital abnormality, that is to say that a child may be born into the world with adenoid growths in its nasopharynx. In order to realise the symptoms that adenoids may produce in young children, our best plan will be to look at the clinical history of some cases which I have had under observation. I shall begin in the first place with the simplest symptoms to which adenoids may give rise in infancy. For instance, a child of five months old was brought to me with the history that it had been snuffing at the nose and had difficulty in breathing since the time she was born. The family history was perfectly good, and there was no suspicion of congenital syphilis. On examination the respiration was laboured, and the lower interspaces were

drawn in during inspiration. The child had also a markedly adenoid facies. On examining the naso-pharynx one found quite easily a mass of fibrous adenoids, and after their removal the child was brought back with almost complete relief from all its symptoms. That would be one of the simplest cases of adenoids in infancy, and you will observe that it occurred in a child as young as five months. I have seen another case in a baby of nine months, which was brought on account of discharge from the nose, accompanied by occasional epistaxis. This child had also a slight bronchitis, which is a very common accompaniment of adenoids, and it had marked vegetations in the naso-pharynx. In that case too the epistaxis and the other symptoms disappeared after the naso-pharynx had been cleared out.

Another group of cases show, as their chief symptom, *cough*. A child only two months old was brought to me on account of a choking cough, which came on in paroxysms. (That is worth noting, it is the typical sort of cough which is associated with adenoids in the naso-pharynx. On account of this cough some of the patients with adenoids are supposed to be suffering from whooping-cough.) Further, the cough in this child was invariably accompanied by the involuntary passage of fæces, owing, I suppose, to the extreme spasm which it caused. Examination of the child showed the lungs to be perfectly healthy, but the naso-pharynx was full of adenoid growths.

Another baby had snoring respiration, and was only able to sleep in snatches, out of which it started up in a condition of partial asphyxia. During the day it also suffered from paroxysms of choking cough, and sweated profusely about the head, which is another symptom not infrequently associated with adenoid growths in infancy. In this case also great and immediate relief followed operation.

Another class of case exhibits a *stridulous respiration* as the chief characteristic. A baby eighteen months old was brought to me for a "catch in the breath," which came on at intervals during the day and was also present during sleep. The veins of the head and scalp were distended, particularly at night, and there were moist sounds in the lungs. The child had enlarged tonsils and adenoids. I subsequently heard that it died

suddenly a day or two after I saw it, and before operation was performed. I have known that occur in another instance, but in the majority of cases one finds that the stridulous respiration is much slighter in degree, and the children after having been submitted to operation make a good recovery.

Another class of case is that in which the child has *difficulty in swallowing*. A child sixteen months old was brought on account of difficulty in swallowing, this difficulty having been observed for a long time. The trouble was found to be greatest in the case of liquids, and on that account the child experienced difficulty in taking the bottle, and had to be fed by means of a spoon, and then the fluid tended to run down the side of the mouth unless the head was held very much back. Examination showed that the tonsils were enlarged, and there was a mass of adenoids in the naso-pharynx. The throat was operated upon, and when the child was seen two months later the mother reported that there was very great improvement. Another child, three years and six months old, had the same difficulty in swallowing, which dated from an attack of measles one year previously. The patient suffered also from deafness. The naso-pharynx was full of adenoids and there was some enlargement of the tonsils. That child has been operated upon, but I do not yet know to what extent the symptoms have been improved.

You will see from a recital of these cases that the symptoms of adenoid growths in infancy may be very various. They may be divided into three groups. First there are those in which the chief complaint is snuffling and nasal discharge, occasionally accompanied by epistaxis. The second group is that in which the chief complaint is dyspnoea, often paroxysmal in character, and the breathing is definitely stridulous, frequently accompanied by attacks of choking cough. The third group comprises those in which the difficulty complained of is associated with swallowing, and especially with the swallowing of liquids.

The presence of adenoids in each of these groups may simulate other medical diseases, and it is on that account that they are particularly interesting to the physician. For instance, where there is nasal discharge, snuffling, and epistaxis, one has to distinguish between adenoids and

congenital syphilis. You know that one of the symptoms of congenital syphilis is snuffling and the discharge of ichorous fluid from the nose, and there may occasionally be a little bleeding. Under such circumstances it is often difficult to tell whether the discharge and snuffling are due to congenital syphilis or to the presence of adenoids in the naso-pharynx. The only way to settle the matter is to make careful inquiry into the family history and to look for other signs of congenital syphilis besides those in the nose. In addition, I think the discharge from the nose due to the presence of adenoids begins sooner after birth than it does from congenital syphilis. You may know that there is a distinct interval between birth and the appearance of the discharge of congenital syphilis, and sometimes that interval amounts to several weeks. But the snuffling and discharge from the presence of adenoids may begin immediately after birth. You might think that a local examination of the naso-pharynx ought to settle the matter, but unfortunately it is difficult to examine the naso-pharynx of children satisfactorily. If any of you have tried it you must have noticed that there is an extremely small space between the soft palate and the posterior wall of the pharynx in a young infant, so that you can hardly get your finger up behind the nose. Therefore one can only infer the presence of adenoids in them from the symptoms; one cannot, as is the case with older children and adults, make certain of it by digital examination. Anything like posterior rhinoscopy, I need hardly say, is quite impossible in young infants.

Another medical disease which may be simulated by these growths is the condition known as *congenital laryngeal stridor*. Some children are born with an inability to breathe properly; they have a stridulous respiration which causes a crowing sound, a sound which is sometimes compared by the mother to that made by a hen. Such stridulous respiration may be very closely simulated by the second group of cases of congenital adenoid growths. I do not know that there is any means of always making absolutely certain which kind it is that one is dealing with. I think it is probable that congenital laryngeal stridor is to be regarded as a neurosis of respiration, the child not having yet learnt to co-ordinate its respiratory muscles. In health the two vocal cords open out when the

child makes an inspiratory effort. What seems to happen in congenital laryngeal stridor is that the opening out of the true vocal cords lags behind the making of the inspiratory effort, so that the child elevates its chest, and does not simultaneously open out its true vocal cords. Then when the air rushes in there is a crowing sound. How the presence of congenital adenoids leads to the stridulous respiration I am not prepared to say, but there can be no doubt whatever that congenital adenoids do lead to that, for not only is it illustrated by the cases I have mentioned, but many other observers have recorded a similar condition. There was a case reported some time ago in the 'Lancet'* by Dr. Eustace Smith, in which the symptoms disappeared immediately after the removal of the adenoids.†

Now we come to the third group, those in which the chief difficulty is that of swallowing. These are an interesting group, as they may lead to serious malnutrition. I have seen cases of marked wasting in young infants brought about by this difficulty in swallowing milk. The child has been unable to suck the bottle, and has had to be fed by the spoon, and that was often found inadequate. The mechanism of these cases is easy to understand. When the naso-pharynx is blocked by a mass of adenoids it is impossible for the child to breathe through the nose while it is sucking. It has only its mouth to both breathe and suck by, and consequently, when it is using its mouth for sucking it cannot be using it for breathing, and it gets short of breath, and thereupon instantly ceases to suck. Accordingly, it gives up the bottle in despair, and all attempts to feed such a child by the bottle end in failure. Even the feeding by means of a spoon in such cases may be difficult because I think the presence of adenoids interferes mechanically with swallowing; that is to say, not merely through the respiration, but by opposing mechanical obstacles to the act of deglutition. One way in which you can imagine that happening is that the mass of adenoids may prevent the elevation of the soft palate into the naso-pharynx, which is a normal occurrence during healthy de-

* March 19th, 1898.

† See also Lack ('J. of Laryngology,' 13, p. 303, 1898); Lubet Barbon ('Revue des Maladies de l'Enfance,' 9, p. 499, 1891); Huber ('Archives of Pediatrics,' xi, 38, 1894); and Coupard (abstract in 'Jahrbuch f. Kinderheilk,' 28, p. 247).

glutition. This difficulty in swallowing may sometimes be extremely marked. I remember a case which was brought to the London Hospital, and was under the care of one of my colleagues there, in which the difficulty was so marked that the child could only swallow when it held its head very far back, like a bird. You have perhaps noticed that when a bird drinks it takes a mouthful, and then holds its head back in order to swallow it. In the case of this child, other children had given it things to drink so as to have the fun of watching it throw its head back in order to swallow them. That child had a considerable growth of adenoids, and the condition very much improved after they were removed.

So much for the occurrence of adenoids in young infants. They may lead to symptoms which it may be difficult to diagnose from those due to other medical diseases, and they are symptoms which may lead to serious impairment of health.

Now when we pass to older children we find the condition is much easier to study, because in their case you can make certain of the existence of adenoids by digital examination. I need hardly remind you of the enormous frequency of adenoids in children up to the age of puberty. After that age they tend to disappear spontaneously. But children at the period of the second dentition suffer from adenoid growths with great frequency, and the danger is lest one should take an exaggerated view of the presence of these growths. Some of you who have attended my out-patients may think I am inclined to over-estimate the importance of adenoids in children and the part which they play in medical diseases, and after the recent pronouncement of such an eminent specialist as Sir Felix Semon, perhaps it requires some little courage to maintain that adenoids produce remote or medical effects at all. Yet I think no one can see much of disease in children without being convinced of the frequency of such a condition, and the fact that it seems to stand in causal relationship to certain remote symptoms in many instances. I can only advance the histories of certain cases in proof of this statement.

Let us consider, first of all, the relationship of adenoids in older children to *diseases of the respiratory system*, because it is upon the respiratory system that the consequences of adenoids most directly fall. Now the first respiratory disease

which it is important you should recognise as a common consequence of adenoids is simple *bronchitis*. You will frequently have children brought to you with severe cough, and on examining the lungs you will find a few moist sounds at the bases. Such children will be found to be mouth breathers and snorers at night. It is not difficult to see how adenoids lead to bronchitis. What is the normal function of the nose? It has been calculated that the nose yields up to the inspired air as much as two quarts of water in twenty-four hours;* it adds that quantity of water vapour to the air which we inspire besides warming it up to the body temperature. It has also been calculated by Dr. StClair Thomson and Dr. Hewlett that 1500 micro-organisms are inspired every hour, and that sometimes this quantity is enormously increased. Yet when you come to examine the mucous membrane of the trachea you find that it is practically sterile, that is to say, the micro-organisms do not get beyond the outer passages. Thus it will be seen that the function of the nose is to moisten the air and to sterilise it, and if the nose is obstructed, as it is in marked adenoids, these two functions are imperfectly performed, and the child is breathing an unduly dry air, which is, moreover, insufficiently sterilised, and the consequence of that may be that the lining membrane of the tubes get irritated, and the child develops bronchitis. If you are going to treat a case of bronchitis of that sort successfully you must first see that the upper air passages are rendered patent.

Another respiratory condition definitely associated with adenoids in certain cases is *asthma*. I recognise that I am here on more dangerous ground, because asthma is one of those reflex neuroses, the occurrence of which is apt to be exaggerated. I shall have to mention one or two reflex neuroses which arise not uncommonly in patients who are the subject of blockage of the nose or naso-pharynx, and one of those I certainly think is asthma. Take these two cases. A girl, æt. 8, was brought to me not long ago with the complaint that she had suffered from asthma since infancy. On examination, the chest was found to be flat and badly developed, and there were a few moist sounds at the base of each lung, as well as an extreme development of adenoids in the naso-

* Mayo Collier, 'Journ. of Rhinology,' January, 1901.

pharynx. The adenoids were removed and no other treatment was adopted, and four months after the operation it was reported that she was perfectly free from the attacks. Another patient was a boy of six years and three months who, since three and a half years of age, had been treated for asthma, but without any real benefit. The mother stated that he had a severe attack of nocturnal dyspnoea lasting for several hours, occurring once a week. He had excessive enlargement of the tonsils and a large mass of adenoids. He also was operated upon without the adoption of other treatment. Three months later the mother reported that he was almost free from attacks except when he had a cold in his head. Without mentioning other cases in which there seemed to be a definite relationship between adenoids and asthma, those that I have referred to are quite conclusive. I admit there are cases of asthma and adenoids in which removal of the adenoids does not seem to cure. That is to say, if you find adenoids and asthma co-existing you cannot promise that removal of the adenoids will invariably cure the asthma. I had a very interesting case illustrating that point. It was that of a child five or six years of age who suffered from asthma. He lived in the country, and had suffered practically all his life from the disease. Unquestionably he also had a large mass of adenoids. He was brought to the hospital here, and when I saw him I recognised the presence of these adenoid growths, and I advised that they should be removed. This was done, and he remained in London several weeks. During that time he was practically free from attacks. Of course I naturally concluded that the operation had cured the asthma, and he went back to his home in the country. I followed the case up, and I found that immediately he got back there the attacks of asthma recurred. This, therefore, seemed to be one of those cases in which the asthma only came on in certain localities, the child having been born in a locality which produced it in him. In London he remained free from attacks. These cases are not infrequent, and you may be easily deceived by them. The mere presence of adenoids does not insure that removal of them will cure the accompanying asthma.

The next system which may be effected by the presence of adenoids is the *alimentary*. Those of you who have seen much diseases of children's,

especially in the out-patient department here, must have been struck by the fact that perhaps the majority of cases of adenoids which one sees in children after the second dentition occur in those who are dyspeptic, and in those who suffer from that particular form of dyspepsia to which Dr. Eustace Smith has given the name of "mucous disease." In some ways that is an unfortunate term, but there is no doubt that it does represent a definite group of cases, and cases which occur with great frequency. These children are usually brought to one with the complaint that they have cough, that they have no appetite, and that they are wasting, and, in addition, they are restless at night and suffer from constipation. One has often seen such patients sent up with the diagnosis of phthisis. If you examine them you will find that they have, in addition to the symptoms which Dr. Eustace Smith described in the stomach and intestines, adenoids in a more or less marked degree. It is difficult to be sure of what the relationship is between adenoids and dyspepsia. Some people go so far as to say that the constant swallowing into the stomach of the mucus secreted by the adenoids maintains a condition of dyspepsia. The only way of settling the matter would be to treat some of those cases by putting the nasopharynx into a healthy condition, and observing what the effect was upon the stomach. That I have not done, but I hope to carry out the treatment on these lines in other cases, with the view of seeing what the relation is between the adenoids on the one hand, and the dyspepsia on the other. But certainly in these cases it is extremely important to recognise the existence of adenoids, because of the very frequent mistake which is made in regarding them as cases of pulmonary tuberculosis.

The next system which may be involved in cases in which adenoids are present is the *nervous system*, and we have a large number of cases of nervous disorders associated with, and I think in many cases causally associated with, adenoids in the naso-pharynx.

To the first group belong cases of general mental dulness or *hebetude*. That is a condition—the name "*aproxia*" has been applied to it—which implies inability to concentrate the attention properly. If you go into any of the special classes which are formed under the London School Board for backward children,

you can pick out at once a large number of cases which suffer from adenoids. The way in which adenoids may produce this mental dulness or backwardness has been much disputed. Some people have supposed that the adenoids interfere with the proper return of lymph from the brain. They believe that the lymphatics passing from the brain are blocked, and that therefore the brain is kept bathed in the product of its own metabolism, and in that way its proper action is interfered with. Perhaps that theory is rather far-fetched. A simpler view is that the mental dulness in these cases is due to inability to hear well, that the pupil never properly hears what the teacher says. Such children are not, strictly speaking, deaf; they hear noises, but they do not adequately distinguish words, particularly if they are at a little distance away; and hence they become backward in their class. Another theory seeking to explain an association between adenoids and this backward condition is that the brain is kept constantly in a state of partial asphyxia from the blood not being sufficiently oxygenated. Whatever the true cause is, there can be no doubt of the great frequency with which one finds this general mental dulness associated with adenoids. In all cases in which you are consulted about a child who is backward at school, examine the naso-pharynx, and at the same time examine the ears. You will find in quite a large proportion of them that the interference with hearing is due to the presence of adenoids, and I can assure you, from my own experience, that many of them have been greatly improved by having the throat cleared out. I have that on the evidence of teachers in those special classes to which I have referred.

Another neurosis which may arise from the presence of adenoids is *night terrors*. It is not quite so easy in this case to explain the association, and the cases are complicated by the fact that in the majority of them there is that form of dyspepsia which I have already spoken of present as well. It is difficult to assign a proper share in producing the night terrors to the dyspepsia and to the adenoids respectively, but I have seen cases in which removal of the adenoids alone produced a great improvement in the night terrors. Rey has recorded* the case of a girl who was operated upon

on five successive occasions for adenoids, and in every case this immediately relieved the night terrors, which only recurred with the recurrence of the adenoids themselves. That is a fairly conclusive case in regard to the association of the two. Certainly in the case of any child who comes under your observation with night terrors you should attend to the state of the naso-pharynx, in addition to treating the general health. The proper way to do this is not to give nerve sedatives, such as bromides and chloral. The necessary thing is to attend to the digestive organs, and particularly to give the patients aperients.

Allied to night terrors is another neurosis, namely, *nocturnal incontinence of urine*. That is a condition which, beyond all doubt, is often *maintained* by the presence of adenoids, if, indeed, it is not actually caused by them. The first case of this sort that I saw was an instructive one, that of a boy in a Home in the east of London, who had suffered from nocturnal incontinence of urine nearly all his life. He was sent to me by the superintendent of the Home to see what could be done for him. I found he was a typical mouth breather, that he snored at night, and that he had a large mass of adenoids and enlarged tonsils. His throat was operated upon, and almost immediately the nocturnal incontinence ceased and did not recur. Similar cases have been recorded in large numbers by various other writers. The relation was first pointed out by Major in 1884, and was confirmed by other German writers subsequently to him, and Grönbech has recorded* twenty-three cases in which adenoids were operated upon, and of which twelve were fully cured, five were improved, two were somewhat benefited, three remained as they were, and one was lost sight of. Huber, in America, found † that out of 427 cases of adenoids sixty-one had incontinence, and in thirty-nine of them removal of the adenoids was followed in twenty-six by complete cure and in eleven by great improvement. So if you have a case of enuresis, certainly adenoids should be looked for, and if they are found they should be removed.

Amongst other and rarer forms of nervous disturbances to which adenoids may give rise one

* 'Jahrb. f. Kinderheilk.', 45, p. 316, 1895.

* 'Arch. f. Laryngol. und Rhinol.', ii, p. 214, 1895.

† Abstract in 'Med. Review,' iii, p. 182, 1900.

is *defect of speech*. I have seen several cases of adenoids associated with difficulty in speech. Usually these cases occur in young children, and in the majority of them there is reason to suppose that the adenoids have been congenital. A child of two years of age was brought to me because it was unable to use words at all. It seemed mentally quite bright and it apparently heard ordinary sounds well enough, and the only thing amiss which could be found was the presence of extensive adenoid growths. Operation produced partial improvement, but one found some time afterwards that the growths had recurred. That child has since been operated upon again, and it will be interesting to follow it up and see what improvement of speech follows the operation. I do not know what the difficulty in the speech is due to in those cases; it may be that the child does not hear words spoken by other people sufficiently clearly to enable it to imitate them.

Another minor neurosis which may accompany adenoids is *headache*. It is usually frontal, and is probably due to interference with the proper ventilation of the air sinuses. Such headache is frequent in adenoids. Dr. Crowley* went through a large number of cases, amounting to some hundreds, which had been operated upon here, and in 55 per cent. of them he found there was a history of headache, which had been cured by operation in about 50 per cent. *Vomiting* has been recorded as one of the rarer forms of neurosis, particularly by Breton.† He describes recurrent vomiting in a child which was cured by removal of adenoids. I have not seen such a case, but the peculiar spasmodic cough which adenoids give rise to frequently ends in retching, if not in actual vomiting. *Torticollis* is another and rarer condition which has been reported to be cured by the removal of adenoids. Gillette records three cases.‡ A still rarer form of neurosis of which I have seen one example only, which seemed to be due to adenoids, is *ataxy* or *staggering*. I remember a child, æt. 3, who was brought to me with the complaint that he was wasting, had attacks of nose bleeding, and was very backward of speech and apparently did not hear well. The child had excessive adenoids, and the typical

adenoid facies, mouth breathing and other symptoms. I did not have the throat operated upon at once. Soon afterwards the child developed ataxy and staggered about as if he were intoxicated. This ataxy became more marked after the lapse of a few days, until eventually he was hardly able to stand at all. He was then taken into the hospital, and on examining him more carefully one found that he had marked indrawing of the tympanic membranes. There was no sign of cerebellar disease, that is to say, no optic neuritis and no vomiting, neither was there any complaint of headache. The case was watched for some time, but did not improve. It was rather puzzling to know what the ataxy could be due to, and one thought it worth while to remove the adenoids, on the theory that the indrawing of the tympanic membrane was affecting the semicircular canals. You know that a person with a large quantity of wax in the ear may suffer from acute staggering, and one thought it might be the same with this child. The adenoids were completely removed, and then the ears were inflated. Improvement began almost at once and continued steadily. The operation was performed nearly a year ago, and when I saw him quite recently he was in practically a perfect state of health, and I was told that there had been no tendency to ataxy since he left the hospital. I do not wish to claim that this was a case of ataxy due to adenoids, but I think it is very suggestive, as the improvement began immediately after operation.

Such, ladies and gentlemen, are some of the medical diseases which may be simulated or caused by adenoids, and the question is as to what degree of importance one should attach to the presence of the adenoids in any given case. And here again I warn you against the dangers of exaggeration. I do not want to say for a moment that all those cases which I have mentioned were directly *caused* by adenoids, but they were all, as such cases very frequently are, *associated with* adenoids; and in many of them, when you remove the adenoids the other conditions clear up. It is perfectly true that you can, in the majority of cases find a predisposing cause to the bronchitis, or night terrors, or whatever it is, as well, yet in many of them one can fairly regard post-nasal growths as an exciting cause, and if you remove the growths the symptoms largely cease.

* 'Pediatrics,' iii, No. 9, 1897.

† 'Rev. mensuelle des Maladies de l'Enfance,' 18, p. 235.

‡ Abstract in 'Pediatrics,' ii, p. 540, 1896.

Into the technique of the operation for the removal of adenoids I do not propose to enter. But one may say a word as to when operation is indicated. I do not wish you to understand that in all cases such as I have described if you find adenoids you should proceed to removal at once. I think it is safer, unless the symptoms are extreme, to try simpler measures first. Mr. Arbuthnot Lane has recommended respiratory exercises in the treatment of these conditions, and has said that if one has recourse to them systematically and persistently operation is in many cases unnecessary. Of such a line of treatment I have had no experience, because in the out-patient department of a hospital it is almost impossible to get it carried out with sufficient thoroughness and persistency to afford a reasonable chance of success. In ordinary out-patient practice I think the best plan is to try, first of all, ordinary cleansing of the nose. Very often one finds that symptoms are not due merely to the adenoids, but to their producing an excessive secretion of mucus, which becomes dry and causes blocking. If you can remove that, the adenoids of themselves may not be sufficient to produce serious symptoms. One of the best things for this is the nasal douche. A solution of borax (one drachm to the pint) is a mild and fairly good solvent of mucus, and this is injected up the nostril and allowed to run out by the mouth, so as to wash out the nasal cavity. In many cases this has resulted in a large degree of improvement. In any case, if you intend to proceed to operation it is well to submit the patient to such treatment before the operation is undertaken. One is less likely to get sepsis afterwards if one has first taken the trouble to see that the nasal passages are reasonably clean.

With regard to the operation itself, you must be prepared occasionally for failure; that is to say, you must be prepared for the possible recurrence of symptoms. When first I directed my attention to this subject I used to think that in many of the cases in which the operation seemed to fail the removal of the adenoids had been incompletely carried out. But now I think that is probably not correct. In many cases I am certain that the adenoids recurred, and I think one may go further and say that the younger the child the more likely such recurrence is to take place, and it is often well to prepare the parents for that, and to tell

them that you may require to perform two or three operations before you get a satisfactory result. It was found by Dr. Crowley that of the cases operated upon here, quite a considerable proportion showed recurrence, although there was no reason to suppose that the operation had not been completely performed. Therefore if one finds that the symptoms are not relieved by the first operation it is not fair to blame the surgeon and allege that he has not done his work properly.

Another cause for apparent failure from operation may be that the nasal passages are abnormal. There is nothing commoner than for adenoids to be associated with some other cause of blocking in the nose, such as enlargement of the inferior turbinated bones, or a disordered condition of the septum, and so on. And of course you cannot expect in these cases that mere removal of obstruction behind will result in much improvement so long as the anterior obstruction still exists. So in some of these cases it may be necessary to attend to the state of the anterior parts as well as to remove the adenoids.

Lastly, though in many cases operation may be expected to cure those cases by itself, you must never forget to treat the underlying condition which is so often present. Adenoids may certainly be regarded as a symptom of ill-health, and whilst treating the local disease, you must attend also to the general condition. These children frequently want tonics of different sorts, particularly iron, and they also require attention to the stomach. Many of them, too, are much benefited by a change of air. In other words, whilst you are attending to the exciting cause of their symptoms you must never forget to give attention also to the predisposing cause, which is usually a delicate condition in the child.

Tuberculosis in the Tracheotomised and Intubated.—M. R. Romme ('*Presse Médicale*') has studied the figures of various observers and has come to the conclusion that intubated children acquire tuberculosis only about one fourth as frequently as those who are tracheotomised. When there can be a choice, therefore, as to the method of operation, he would prefer intubation.—*New York Medical Journal*, January 4th.

THE TREATMENT OF HEART DISEASE.*

By G. W. EUSTACE, M.A., M.D.

(Concluded from p. 224.)

WE will now turn to the consideration of the treatment of the second, or stage of mechanical failure of the heart. As a rule, when the heart shows signs of failure dilatation is present, and steps should at once be taken to lessen and, if possible, remove it, firstly, by relieving the heart of all unnecessary work, and secondly, by strengthening its action.

On the degree of failure present will depend the amount of work which may be safely performed; but in all cases of cardiac failure, whether slight or severe, the more complete the rest the sooner will restoration to normal action result, and the less will be the damage left behind; and it is important to remember that on the amount of this damage, which is usually directly proportional to the rapidity of restoration to normal action, will depend the patient's liability to other attacks of cardiac breakdown in the future.

By the term rest I do not merely mean going to bed, but rest as applied to the heart, and to any organ whose increased action reacts injuriously on the heart. To promote this condition of rest of the heart it is equally important that the organs and tissues of the body, whose inactivity increases the work of the heart, should be stimulated to normal or increased action. Here, then, we have the *rationale* of the first step in the treatment of cardiac failure, namely, to diminish by suitable sedatives the activity of the excited and exhausted heart and central nervous system, vainly trying to accomplish more than it can, and to arouse by suitable stimulants the inactive skin, liver, bowels, and kidneys. The strain on the heart being then the first point to relieve, the determination of the site of the strain is important, because this will indicate the correct selection of the means to be employed.

If it be the right side of the heart, which, by its heaving ventricle, distended jugulars, swollen and tender liver, proclaims its fast failing ability to drive the blood on through the lungs, venesection

to the extent of eight or twelve ounces of blood, or the application of ten or twelve leeches over the liver, will rapidly relieve the state of engorgement; but if the site of the strain is the left ventricle, struggling against increased peripheral resistance, these very measures will afford no relief, but only the reverse, while vaso dilators, such as nitroglycerine, will bring about the desired result. Other means to diminish the strain on the heart will consist of mercurial purgatives, followed by diuretics and drugs, such as acetate of ammonia, to increase the activity of the skin.

To restore the central nervous system, which has become exhausted from want of sleep, and is thus injuriously reacting on the heart, the hypodermic injection of morphine and atropine, or bromide, sulphonal, or trional will be employed, and the hurried, irregular, inefficient contractions of the heart met by the digitalis class of drugs.

Such are, in brief, the chief means at our command with which to meet the condition of failing heart; the order in which they are employed is not immaterial.

It may be laid down as a general rule that in severe cases the following sequence should be observed:—To first employ the withdrawal of blood from the system when the indications for such are clearly present, to administer at the same time a mercurial purgative, followed by the hypodermic injection of morphine or dose of sulphonal to cause sleep, which the withdrawal of blood will have gone a long way to induce, and to begin the administration of diuretics and heart medicines when the patient awakes rested and refreshed. To this rule there are, of course, exceptions, as when an excessive degree of ascites exists, or a large quantity of fluid is present in one of the pleural sacs, or the condition is one of asystole, when it is important to quickly rouse the heart to more forcible action. In the first two exceptions mentioned, the removal of the fluid by tapping will take the place assigned to the withdrawal of blood, and will often be followed by as striking results, while in the last it is doubtful if any treatment is of more than temporary avail.

When symptoms of cardiac failure arise in mitral disease, dilatation is leading to engorgement of the right side of the heart; treatment will be directed to relieving the backward pressure on the lines already laid down, and it is in engorged right

* Delivered before the West Sussex District of the South Eastern Branch of the British Medical Association.

heart that the abstraction of blood is often so beneficial. In both forms of mitral disease with cardiac failure, the digitalis class of drugs find their chief opportunity; but as soon as compensation is restored, their use must be discontinued, and this is the more important in mitral stenosis. In cardiac failure when aortic disease is present, the line of treatment will depend on the state of the mitral orifice; if the symptoms are those of failing right heart, we have to deal with mitral symptoms with aortic signs, and the treatment is the same as that just given for failure with mitral disease, differing, however, in that abstraction of blood is very rarely advisable.

In aortic disease with failing left ventricle, and without signs of embarrassed right heart, the condition is very serious, and treatment will have for its main object the lowering of arterial tension, if such is excessive, by the vaso dilators, and direct stimulation of the heart by alcohol, ammonia, and strychnine. Digitalis is here specially contraindicated on account of the increase in peripheral resistance which it causes, and which in aortic stenosis only further adds to the strain on the ventricle, and because, by prolonging the diastole, it aids the backward flow into the ventricle, which in aortic regurgitation is already excessive.

The treatment of certain symptoms in heart failure requires special mention.

Sleeplessness, when accompanied by dyspnoea and restlessness, is best met by the hypodermic injection of morphine, $\frac{1}{8}$ th or even $\frac{1}{16}$ th or $\frac{1}{32}$ th of a grain often acting like a charm. When the condition is less acute sulphonal or trional should be given, bromides are rarely of use, alcohol is uncertain, and chloral positively dangerous.

For dyspnoea, dry cupping, or dependence of the lower extremities by allowing the patient to sit in a chair, will often give relief, but if the difficulty of breathing becomes distressing, nothing acts so well as the hypodermic of morphine.

Pain is rarely complained of, and when present is frequently due to flatulence, which will be greatly relieved by a carminative draught. When pain is of an anginoid character, nitrate of amyl, nitroglycerine, ether and ammonia, strychnine, hot brandy and water, and turpentine stupes will be employed, and if unsuccessful the hypodermic of morphine will again come to our assistance.

Dropsy of the pleural sac or of the peritoneum

will, if excessive, be removed by tapping, and recurrent ascites treated by opium after the method of Graves. Great oedema of the lower limbs or scrotum may assume such proportions as to require draining by puncture, care being taken to observe strict asepsis.

Hæmorrhage rarely gives rise to trouble; it invariably means an overloaded right heart, the treatment of which has already been mentioned.

In the treatment of heart disease success will often depend not only on recognising the indication for a certain line of treatment, but also on knowing how to administer it. Allusion has already been made to the sequence to be observed in the use of the chief classes of remedies at our disposal; a great deal can also be effected by knowing what combinations of drugs to employ, or when and how to use them separately; thus morphine, given hypodermically, will cause less reaction when given with atropine than when given alone. Sulphonal, on account of its slow absorption by the stomach, acts best when given half an hour or an hour before bedtime, and its absorption is hastened by giving with it some hot drink, and it is a well-established fact that if given on two successive nights it often not only fails to cause sleep during the second night, but sometimes increases the wakefulness.

Digitalis is more used, and its use more often abused, than any other drug in the treatment of heart disease. The indications for its use are "frequency, weakness, and irregularity of the pulse, and oedema of the extremities, with scanty, turbid concentrated urine; when these are absent, it is rarely of service."—Broadbent. It always acts better when preceded by a mercurial purgative, and in cases of engorged right heart with oedema, its combination with squill and calomel in small and repeated doses, with pulv. opii added, if looseness of the bowels become troublesome, often acts like magic, while in giddiness and palpitation, the result of slight dilatation of the left ventricle, its administration with the aromatic spirit and bromide of ammonia will answer far better than when given alone, whilst sometimes its best effect is produced by giving a single large dose each night in rectified spirit.

Strophanthus will sometimes succeed when digitalis fails, especially when the latter is not well borne by the stomach; but I have never seen

benefit follow the use of spartein, convallaria, or cactina, given with the same object.

In strychnine we have one of our most valuable remedies; it may be given with digitalis when the latter is indicated, or with iron and quinine as a general tonic; but its greatest use is in maintaining vigorous action of the heart in chronic progressive aortic disease occurring after middle life; it is best given alone, two or three times a day, in doses of two to five minims of the liquor, or the same dose of the tincture of nux vomica.

Iodide of potash is specially indicated when the chronic change occurring is due to syphilis, and its action is increased by giving the aromatic spirit of ammonia with it.

Diuretics are very useful when œdema is present, and a combination of several diuretics will often succeed when the administration of a single one fails.

Belladonna is loudly praised by many, but in my experience it has only given benefit when given at bedtime to lessen disturbance from too frequent micturition.

In coffee we have, perhaps, a more powerful cardiac stimulant than alcohol, and a small cup of black coffee, given at bedtime, either alone, or with ten drops of tincture of digitalis or strophanthus, will often prove of the greatest service.

Alcohol is much more often prescribed than the indications for its use warrant; it is rarely required in the stage of inflammation, except when cardiac failure threatens; its use is contra-indicated when compensation is fully established, owing to its tendency to retard assimilation and excretion, and to induce degenerative changes. It is, however, invariably required during the stage of mechanical failure, particularly when the action of the left ventricle is weak and inefficient, but in cases apparently requiring prolonged administration, it is always wise, after a time, to try the effect of diminishing the amount ordered, when it is often surprising, but by no means unusual, to see much benefit follow. It is best given in the form of whisky or brandy, in measured doses, at stated intervals; in some cases a dose at bedtime has a decided influence in procuring sleep; but when it flushes the face, and increases the rapidity without increasing the force of the heart-beat, it only does harm.

Food should always be nutritious and easily

assimilated, taken at regular hours without hurry, and overloading the stomach, especially before retiring to rest, should be carefully avoided.

The importance of rest has already been insisted on, and in some cases, particularly of dilatation with but little hypertrophy, complete rest for weeks, or even months, will accomplish more than the most careful combination of drugs.

Until compensation is fully established the amount of exercise taken will be necessarily limited, but if judiciously watched, it will generally help convalescence; the best indication is the state of the breathing resulting, hurried or difficult respiration being always the signal to rest.

When an attack of cardiac failure has been recovered from, it is all-important to prevent, or at least postpone for as long as possible, its recurrence. The patient must understand that his heart, having once failed, is very liable to do so again, and that the slightest symptoms of failure must at once be treated, and that his health can only be maintained by sacrificing whatever reacts injuriously on his heart; henceforth he must lead a simple life, comprising the absence of all excesses, simple, suitable food, regular hours, regular exercise, regular sleep, and last, but not least important, insure regular action of the bowels.

I have already occupied too much of your time, and the interesting subject of the treatment of functional diseases of the heart and that of the condition known as angina pectoris must be left for a future occasion.

We have received from Longmans, Green, and Co. the third edition of 'Quain's Dictionary of Medicine.' The work has been largely rewritten and revised throughout, and Dr. Montague Murray, of Charing Cross Hospital, has ably fulfilled the arduous duties of editing this important volume. The recent and ever-increasing incursions of surgery into the realm of medicine have received adequate recognition, and the book is what is claimed for it—a reliable and readily available work of reference for the practitioner and student of medicine. We shall hope to be able, in due course, to give it a more adequate notice.

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A CLINICAL LECTURE ON OPERATION FOR THE RADICAL CURE OF INGUINAL HERNIA AND ITS RESULTS.

Delivered at St. George's Hospital, December 17th, 1901.
By G. R. TURNER, F.R.C.S.

I WANT first of all to show you a fireman on whom I operated for hernia some years ago. He has never been troubled at all since the operation, and he carries on his work as a fireman like any ordinary man. There is no yielding of abdominal parietes, and by putting your finger over the scar you would hardly know that operation had ever been done. If you saw such a case some weeks after operation you would feel a puckered hardness underneath the cicatrix, but in the older cases this is no longer noticeable. I thought I would show you one of these patients at the lecture. There have been numbers of them in the male surgery lately who have come up to show themselves in response to my inquiry as to the result of the operation for radical cure of hernia.

As you will have inferred from my showing this case, the subject I wish to bring before you this afternoon is that of the radical cure of inguinal hernia. I also wish to bring to your notice a number of my cases into whose present condition I, by the kind help of my house surgeon, Mr. Jones, have inquired recently; that is to say, those cases I have operated upon in St. George's Hospital. There is a good deal of room for legitimate difference of opinion as to what cases are suitable for operation and as to what operation is the best, as well as with regard to the results that have been obtained by any of these operations.

First of all I should like to describe, in more or less detail, the operation which I do myself. As you are probably aware, these open operations for

the radical cure of hernia were originated by Sir W. Mitchell Banks. To him is due the treatment of this condition by the open rather than by the subcutaneous method, which was the only one in vogue in the days of my studentship. In other words, Mitchell Banks must be regarded as the father of the open operation for hernia. But of course it is due to Lister that we are able to do it at all, because asepsis is one of the necessary conditions of operation in this way.

The operation which I have done in all the cases which I shall bring before you, some 200 in number, that is to say 135 at St. George's Hospital and the rest elsewhere, for instance, at the Seamen's Hospital and in private, is a modification of the well-known Bassini operation. It differs from the Bassini operation inasmuch that the cord is not handled so much, and is placed in a different situation. In Bassini's operation the cord is placed superficial to the conjoined tendon of the internal oblique and the transversalis, between it and the external oblique. The modification I do is that the cord is placed underneath the conjoined tendon of the internal oblique and the transversalis, which is brought down and stitched to Poupart's ligament in the same way as in Bassini's. But Bassini, at any rate in his first cases, used silk as his buried suture. Now I do not believe in silk as a buried suture; I have seen so many cases in which silk has made its way out, and I, therefore, in practically all my cases, use kangaroo tendon. Coley, of New York, has drawn attention to the troublesome sinuses which have followed the use of silk as a buried suture. Such are the main differences between Bassini's operation and the one I practise. Every surgeon, I suppose, has some modification, and some of these that are done here by my colleagues I will mention presently. There are various points in the operation I should like to dwell upon as I go along.

First of all with regard to the incision. One makes the incision from practically the external abdominal ring, about two and a half inches to three inches upwards and outwards, a good inch above Poupart's ligament, and parallel with it. There is a little point worth remembering in making the incision rather at a distance from Poupart's ligament, it is this: that in the completed operation, when you have stitched the conjoined tendon of the internal oblique and

transversalis to Poupart's ligament, those deep sutures would not correspond with your line of superficial sutures through the external oblique. You can see this in these diagrams.

This is the same in Bassini's operation. Having made the incision through the integument, the first things to meet are the little superficial blood-vessels which come out at the saphenous opening and run up on the abdominal wall. These are vessels which should give no trouble as far as bleeding is concerned, but I hold that it is very important that you should at once clip and, if necessary, ligature them, and for this reason: that if you do not occlude them you are bothered by the blood that comes from them. This blood stains the deeper tissues so that you may not later on readily distinguish what is external oblique, what is sac, and what is cremaster muscle. It is for this reason I take the trouble to make the operation as far as possible a bloodless one. Having come down to the external abdominal ring, I insert my forefinger, usually of the left hand, into it, and in that way pull up some fibres of the external oblique. Then with a knife I divide the external oblique at a little distance from the external abdominal ring, and separate with a director the aponeurosis of the external oblique from the structures underneath, so that I can clip each of its edges. I put a clip on the aponeurosis, and use those clips in the later stages of the operation as retractors. They are most useful when one is passing the deep stitches which eventually will suture the conjoined tendon to Poupart's ligament. Having divided the external oblique and clipped its margins, I go through the external abdominal ring and divide all the external spermatic fascia which comes from the margins of the ring and forms one of the coverings of the hernia. Then I free the aponeurosis of the external oblique from the conjoined tendon above, and expose Poupart's ligament below, and in that way I have the two structures which I shall afterwards suture together well defined. What one now sees is the sac of the hernia covered by the cremaster muscle, which varies very considerably in different individuals, coming down through the external abdominal ring which has been laid open in the manner described. The next step in the operation is to go through the fibres of the cremaster muscle, and this should be done in all cases very carefully and completely in

the same line as the external oblique. The structure which you now come upon is the infundibuliform process of the transversalis fascia. Dividing this you can define the sac of the hernia. The sac is then peritoneum, and peritoneum alone. It is most important for the ease with which you subsequently separate the sac of the hernia from the cord that you should divide deliberately all the superficial structures as I have done in removing the specimen of sac which I pass round. There should be peritoneum, and peritoneum alone. If you once get down to the peritoneal sac of the hernia, with peritoneum on all sides, then the separation of the sac from the cord, which is the next step in the operation, is comparatively easy. Having then defined the sac one takes hold of it and proceeds to separate it from the cord. Sometimes it may be a little difficult to exactly find the sac in bubonocoeles; indeed, cases are on record where no sac has been found and the operation has been abandoned. I am accustomed to look for its white edge. When once you find that, the subsequent separation of the sac from the tissues of the cord is comparatively simple. As far as the sac is concerned, personally, I remove the whole of it, as in this preparation passed round. I do not leave any of it *in situ*, as some surgeons recommend. I remove the whole sac and trace it up to the internal abdominal ring, reducing any contents that there may be in it, and dividing and separating any adhesions that there may be between the sac and the contents, because very often one finds adherent omentum. I then ligature the neck of the sac with catgut. Some surgeons employ not only a ligature to the neck of the sac, but they twist the sac before they ligature it. I do not do this, nor do I deal with the sac by stitching it in any abnormal situation. I simply tie the neck with catgut, tying the string on a finger of an assistant inside the sac, which prevents any protrusion of hernia at the moment of ligature. Having removed the sac and separated it from the cord—and, of course, in congenital hernia where the sac goes down to the testis that separation of the sac is more difficult than in the acquired form—one now deals with the conjoined tendon and brings that down and stitches it to Poupart's ligament. In passing the stitches through Poupart's ligament there are various little points which are of use. Put the first stitch through Poupart's liga-

ment, where it is connected to the top of the pubic bone. After having put a stitch through here, if you pull on it you will very easily define the rest of Poupart's ligament, and pass the stitches, usually three in number, sometimes four, pulling on each one as you pass the next. You will in this way pull Poupart's ligament from the deep vessels, such as the femoral artery and vein, and run no risk of wounding them. Having passed the stitches through Poupart's ligament, I clip each one of them, so that I know which part of the suture I shall want to bring through the conjoined tendon. I next take my curved needle on a handle (unarmed) and pass it through the conjoined tendon in three separate places, threading it each time with the Poupart ligament stitch that I want, and so tie the conjoined tendon down to Poupart's ligament. If that is done systematically from within outwards, you can obliterate the inguinal canal, that is to say, you fill in the space bounded above by the conjoined tendon below, by Poupart's ligament. In describing this operation you will notice I have said nothing about the cord. One has not handled the cord except to separate the sac from it. I do not touch the cord after I have separated the sac from it. I stitch the conjoined tendon to Poupart's ligament in front of the cremaster muscle, so that the cord lies behind the conjoined tendon of the internal oblique and transversalis, which is stitched to Poupart's ligament, between that and the peritoneum behind, not, as in Bassini's operation, in front of the conjoined tendon. I irrigate the wound once or twice during the performance of the operation with 1-in-40 carbolic, and then I stitch up the external oblique by continuous kangaroo tendon stitch. Very often I have used interrupted sutures. In stitching up the external abdominal ring there is one little caution I should like to give you, and that is not to stitch it up so tightly that you compress the cord. In one of my earlier cases I did that to the cord on the right side. I stitched up the ring just a little too tightly, with the result that I gave my patient a right varicocele. Otherwise he was cured of his hernia. I do not believe he was aware he had the varicocele, but that is a point to avoid. You must leave sufficient room for the cord to pass into the scrotum without producing any constriction. The points in the operation which I wish to draw attention to are as follows:

One avoids all interference with the scrotal tissues; the operation is made as bloodless as possible; the conjoined tendon is stitched to Poupart's ligament in front of the cord rather than behind it, as Bassini does, and one uses kangaroo tendon in preference to silk for the buried sutures; no veins are removed. I am sure from my experience at the Convalescent Hospital at Wimbledon that buried silk sutures constantly come away. Kangaroo stitches come away occasionally if there is anything like sepsis in the wound. In the vast majority of cases, however, kangaroo tendon remains in its place and does its work well. I am aware that a great authority on this subject, Sir William Mitchell Banks, says he believes that unless you raw the edges it is no good stitching the conjoined tendon to Poupart's ligament; he says, indeed, that he would just as soon stitch the edges of a cleft palate together without previously preparing the edges as far as union is concerned. You may or may not get union between the conjoined tendon and Poupart's ligament, but the new arrangement of the muscle lasts sufficiently long. It has been urged against operations involving division of the external oblique that there is a greater tendency to yielding, and that there is a tendency to a form of ventral hernia greater than in those cases where the pillars of the ring are alone dealt with; but I cannot think that this is really so if only the arched fibres of the conjoined tendon are properly stitched to Poupart's ligament, and the inguinal canal in this way thoroughly obliterated. It is one of the *loci communes* of surgery that in old hernia the inguinal canal ceases to be oblique and that the internal ring is nearly straight behind the external one, that, in fact, there is very little inguinal canal. This, no doubt, is so in old cases, but it does not affect the importance of this suture of the internal oblique and transversalis to Poupart's ligament in preventing subsequent yielding of the abdominal parietes. If a case is seen some time after operation, a hard rigid band corresponding to the line of suture can be felt, which seems, at any rate, to be quite one of the strongest parts of the abdominal wall. I would myself compare the operation of merely suturing the external ring to the shutting of a door, that of obliterating the inguinal canal to the blocking up of a room. One prominent Australian surgeon, who has had considerable experience of operations

for radical cure, regards it as essential that the sac should be removed flush with the peritoneum, and he says that is all that is wanted. He also regards the conjoined tendon of the internal oblique and the transversalis as possessing certain sphincter powers which prevent the descent of the hernia. Well, that may or it may not be so. But I cannot help thinking that when there has been an alteration in the anatomy of the conjoined tendon, when it is brought down so as to be in contact with Poupart's ligament, there is much less likelihood of hernia occurring subsequently than when it is left in its normal position.

Some surgeons advocate torsion as well as ligation of the sac. It is my practice always to remove the whole of the sac unless some extensive adhesion of the bowel makes it more desirable to return the bowel with the adherent portion of the sac into the abdomen. Long-continued manipulation of adherent sac involving prolonged dissection and handling of the bowel undoubtedly increases the gravity of the operation, and has appeared ere now to lead to fatal results. I have not myself practised leaving the distal part of the sac *in situ*, except in one case of a very fat, middle-aged man, with a large hernia and a considerable quantity of adherent gut and omentum. This patient I thought a very bad subject for operation, though, as a matter of fact, his wound healed by first intention. It would be interesting to know whether the distal part of the sac when left over gives trouble by being the seat of hydrocele or hæmatocele. I have no personal experience of either Macewen's or Stanmore Bishop's methods of suture and invagination of sac, or, indeed, of Kocher's operation, but I have seen the latter practised and shall not myself attempt it.

Nearly every one is agreed that one of the great points in operating for the radical cure of hernia is to ligature the sac flush with the peritoneum, and surely this can be best done when the parts are fully exposed. In some cases omentum is adherent to sac right up to the internal ring, or there may be processes of subperitoneal fat in the canal. Can cases be as adequately treated through an enlarged external ring? I think there is too much fear of this slitting up of external oblique. If suture of the enlarged external ring is going to succeed, why should suture of the same structure a little external to the ring fail? In one of my

hospital cases, owing to some accidental septic contamination, there was acute cellulitis, and some sloughing of the cellular tissue and external oblique aponeurosis. The stitches connecting the arched internal oblique and transversalis to Poupart's ligament held, and that man, except that his scar was slightly more depressed and adherent, had been going about without a truss, and with his hernia as radically cured as any of those I have seen, until, some two and a half years later, he was killed in a railway accident. With regard to the wearing of trusses after operation, in my opinion, if that is necessary, there has been no radical cure. Surely a truss, by its pressure, must do little but harm. In some exceptional cases operation must have for its object the wearing of a truss by the patient unable previously to do so, but these cases in which some local or general condition of the patient forbids a true radical cure should be rare.

With the kind assistance of Mr. Jones, my house surgeon, I have lately been inquiring into the results of the operations which I have performed here for this condition. My own experience of the operation I have described to you is about two hundred cases. At first I thought the number was greater, but that is about the number which it works out at. One hundred and thirty-five of those cases have been at St. George's Hospital, and it is these latter I shall refer to chiefly to-day. The others were done at the Seamen's Hospital and elsewhere. Of course a sailor is a migratory bird, and one very soon loses sight of him when he goes away. I may say, however, with regard to the Seamen's Hospital, that one of the Sisters, who naturally saw a good many of the patients when they revisited the hospital, told she had not known of a recurrence amongst my cases. Of course that does not prove that there were no relapses, and I have only that amount of evidence with regard to the sailors.

I have had one death in these 200 cases, and that was a surgical calamity. It was that of a young man with a congenital hernia who died from sepsis and sloughing of the cellular tissue of the abdominal wall. The inflammation extended eventually to the peritoneum, and he died on the eighth day. This patient and another in which there was some sloughing of the aponeurosis of the external oblique, were operated upon one after the

other on the same day, and I should think that some dirty instrument, swab, or sponge must have been used. There was no special difficulty in either of the cases. Both had cellulitis, both sloughed, and one died. That was a most deplorable accident, but it was one which might have followed any kind of operation where instruments are used in common by many surgeons and on all sorts of cases. That such calamities are so very rare in a hospital like St. George's, where such a multitude of operations are performed, is testimony to the care and vigilance of those in charge of theatre and the sterilisation of the instruments. I have operated on one case three times, under the following circumstances:—The original operation promised well. The wound healed by first intention, and after a week the man was sent to the Convalescent Hospital at Wimbledon, with orders to rest in bed. Instead of that, he walked about the hospital grounds, and re-ruptured himself; at all events, the stitches gave way, leaving a ventral hernia. At the second operation some buried silk sutures caused some irritation and came away. The man was all right for some months, but returned to me two years ago with some yielding of the abdominal parietes. I saw him the other day, and he is now, by my direction, wearing a truss, and has no hernia. His ring, however, is nearly the size of a shilling. But for his folly in disobeying orders, and walking about at the end of a week, he would doubtless have done perfectly well. Another case which I am not altogether satisfied with was that of a German who had been operated upon elsewhere—I do not know by what method—but without success. He had a large ventral hernia when I first saw him; and although his condition was ameliorated by operation, the result was not such as to enable him to do without artificial support. These are the failures I know of in hospital.

I have had only one relapse after this operation in private, and that occurred in a little boy, who for three years after the operation had been performed had been perfectly well, but at the date I mention lifted his sister, and was supposed to have re-ruptured himself. When I saw him there was a little bulging at the outer edge of the scar, which I thought it would be right and proper to remedy. This I accordingly did. When I cut down I did not find any new sac coming down. There was only some yielding of the parts, which I

corrected by the insertion of kangaroo stitches. Since this second operation, which I did two years ago, he remains perfectly well.

The methods employed by my colleagues in operating at St. George's have been as follows :

One of our surgeons tells me he nearly always performs Bassini's operation. Another isolates the sac, stitches up its neck with a purse-string stitch, leaves the lower part *in situ*, and sutures the external abdominal ring. In one or two cases, where the ring has been large enough to allow it, he sutures the inguinal canal. He occasionally uses Macewen's method of dealing with the sac. Another surgeon, in the case of children, ligatures the sac and closes the rings. In adults he invaginates the sac, and does a modified Macewen's operation ; or, if the size is great, and the parts thin, he does a Bassini or Halstead operation. Sometimes he does a Kocher, and sometimes he sutures the conjoined tendon to Poupart's ligament. Another surgeon usually does a modification of Bassini's operation, ligaturing the neck of the sack and dividing it. The ends of the sac are tied by him into the conjoined tendon, but not crumpled up, as by Macewen. Lately, however, this surgeon has discarded making the cord superficial. Another surgeon usually does a modification of Bassini's operation, performing torsion of the sac before ligaturing it. In his incision he avoids the scrotal tissues. He uses silk buried sutures, and passes them according to Macewen's directions. He does not make a very complete division of the external oblique. Other surgeons in the hospital perform the same operation as I do.

Now on what cases should a surgeon operate ? Obviously not on very young children, who can, and do, grow out of their rupture if it is efficiently treated by a truss. But there are some cases, even in young children, where the hernia is large and uncontrollable, where it is practically certain that truss treatment, requiring as it does constant care and skilled supervision, will fail. I should not hesitate to recommend operation in all cases in children above the age of five or six when they have been wearing a truss, and in all cases of young adults and healthy middle-aged men. I would let no young man—except for very special reasons—be condemned to wear a truss, that is to say, I recommend operation even for bubonocoele. This class of case should always give a good

result ; no complications are likely to be encountered, and if left to a truss the rupture at any time may become scrotal or even strangulated. It is astonishing what a long sac a bubonocoele may possess. I have found one, not of the congenital variety, as long as my middle finger, in which the patient distinctly stated the rupture never was scrotal. Surgeons who do not operate for bubonocoele can have no real confidence in their radical cures, yet they reserve the operation for difficult cases in which complications are likely to be met with. In this they seem to me to resemble those surgeons who many years ago were aghast at Lister wiring a recent case of fractured patella, and yet did not hesitate to try their hands on the much more difficult old-standing cases. That operation is not suited for all cases is obvious. It must be formidable in cases of large hernia with much irreducible contents, especially if there are adherent intestines, and yet these are the cases where there is constant risk of strangulation. By the way, it should be remembered that this adhesion is generally due to the wearing of a truss on a hernia that is down. Old, debilitated, or alcoholic subjects with renal disease are ill-suited to the handling inseparable from these operations. Skin disease, such as eczema, or any local trouble, such as enlarged glands, should be remedied before operation is undertaken. Indeed, if there is reason to believe that, from any local or general cause, union by first intention will not take place, in my judgment the operation, except in the case of strangulation, should not be undertaken.

This last remark leads me to the consideration of strangulated inguinal hernia and the operation for radical cure. If a modified Bassini's operation such as I have been describing is performed on these cases nothing more is required. The surgeon rarely has to use the hernia director or the hernia knife. By doing this modified Bassini for strangulated hernia you save all such dangers as reduction *en masse* ; there is no deep diving with a hernia knife to divide constricting bands ; you simply operate on the case just as if you were going to do the radical cure. You see the contents of the sac and deal with them on ordinary surgical principles. You can do the radical cure in the same time as it takes you to operate upon an ordinary hernia. With regard to this question of time, this is an operation which anyone who is

familiar with it can do quickly. Last Thursday I did two cases on one individual, that is to say, a double case. I was fourteen minutes doing the first operation and fifteen minutes the second, thus doing both operations within half an hour. That, of course, was a simple uncomplicated case. If the case is complicated it may take half an hour or even longer to do one side.

With regard to the contents of the sac, in two of my cases the appendix, perfectly healthy, was present, and I resisted the temptation to remove it. In one of these it was the sole occupant at the time of operation, though at other times the hernia had been large. I have never met the bladder, but have come across a malformed uterus and two ovaries in one sac. In another case, that of an hermaphrodite, I found a rudimentary testicle. In this case the external genitals were perfectly feminine. The vagina ended in a *cul-de-sac* an inch long. I found two rounded loose bodies (probably derived from appendices epiploicæ) in one case. In one or two cases I have removed enlarged glands at the time of operation, and in, I think, two cases, I have removed them some weeks before. In some few cases where union has not occurred by first intention there has been a history of old syphilis. These cases, however, have granulated up readily.

Now with regard to results, I have, as I have already mentioned, had one death. I pass round a table of my results of 101 cases operated on here. More recent cases, although known so far to be well, have, for obvious reasons, been excluded; sufficient time has not yet elapsed to allow of their being classed as cured. Curiously enough, it is amongst these recent cases that we have seen the only relapse. It is that of a man operated upon last March. The kangaroo tendons came away, and there was some suppuration, so that he was in the hospital seven weeks instead of the usual three. The hernia recurred about six months afterwards. We know of good results in 70 out of the 101 cases. Seven of these have written and the rest have been up to the hospital to show themselves. We have been unable to trace twenty-five. The rest are well, but their operations are of recent date. One other case suffered from acute bronchitis shortly after leaving the hospital, and in all probability it is another case of relapse. Though written to repeatedly

and living in London he has not been up to show himself. Of the twelve strangulated cases, all of whom recovered, we have been unable to trace three. The rest are well and apparently cured. None of these cases have worn or are wearing a truss. All sorts of occupations are represented—porters, painters, coal porters, labourers, costermongers, firemen, scaffolders, servants, carmen, hammermen, and a rough rider in South Africa. Many whom I have recently seen have told me that they have been in the habit of lifting heavy weights since the operation. Of my results at the Seamen's Hospital I am unable to give statistics for the reasons I have already given.

I have looked up our records here for the last ten years and the following is a table of them. The operations have been performed by nine surgeons.

Operations for Radical Cure of Hernia from 1892 to 1901.

	Inguinal.	Femoral.	Umbilical.
1892 .	11	1	—
1893 .	18 (1 died)	1	—
1894 .	36	2	—
1895 .	53 (3 „)	2	—
1896 .	54 (1 „)	3	1
1897 .	59 (2 „)	6	—
1898 .	65	6	1
1899 .	68	7	1
1900 .	71	6	3 (1 died)
1901 .	101	4	2
	536	38	8 (1 died)
In hospital .	4		
	540 (7 died)		

The last 320 consecutive cases show no deaths.

In 540 cases of inguinal hernia there have been seven deaths. I have already alluded to my own case, a surgical calamity due to septic poisoning. Those which have occurred amongst my colleagues are as follows :—One surgeon tells me his case was due to a dirty sponge. He, like myself, operated upon two patients in one day; both had erysipelas, and the hernia case died. A third case, in which he used swabs, was not affected. Another surgeon's case was a child sixteen months old. The sac was difficult to find. The wound healed, but broke down again and sloughed. The child had convulsions and empyema, which was opened on the right side. The death was due to broncho-pneumonia and double pleurisy. It was probably

a septic case. In the practice of another surgeon there were three deaths. The first was in the case of a man *æt.* 66, who had a large irreducible hernia. The colon was adherent to the sac. He died sixteen days after the operation. The bowels were afterwards found to be greatly distended, except one part, which was collapsed and adherent to the wound. There was no obstruction to liquid *fæces*, however, and there was no peritonitis. The second case, in the practice of the same surgeon, was that of a man *æt.* 42. He had a large scrotal hernia with adherent omentum. This was ligatured and removed, the sac dissected out and excised. There was free bleeding from the drainage tube. He died on the tenth day after operation. The third was that of a man *æt.* 52 who had a large scrotal irreducible hernia and hydrocele, which was reduced with difficulty after the ring had been freely excised. He suffered from meteorism in spite of enemata. The wound healed. He turned on his side to go to sleep after being fed, and was found dead. No post-mortem was made. Another surgeon's case had been the subject of strangulated hernia five years previously. He was operated upon, but the bowel was adherent to the sac, and was not returned. A short time before he was under another surgeon's care for strangulation. This was treated successfully by taxis. At the time of the operation for radical cure it was found that the gut was very adherent. It, with the adherent part of the sac, was returned into the abdomen, and the rings were stitched up. The patient died with symptoms of paralysis of the bowel. The surgeon in question tells me that if he met with a similar case he would resect the adherent part. An attempt was made to separate the adhesions before reducing the bowel.

Such are the statistics at St. George's Hospital, where nearly all the various methods of operating have been tried. All my 135 cases there have been done by the modified Bassini's operation, which I have described to you; and personally I would recommend no operation which does not deal thoroughly with the inguinal canal, and that cannot, I contend, be efficiently dealt with unless the aponeurosis of the external oblique be divided. If to these statistics of St. George's Hospital we add some published by Mitchell Banks, Macewen, Hælstead, Bassini, Coley, and the Brothers Mayo, we get a total of 1639 cases, with fifteen deaths.

The mortality from the operation in the past, therefore, has not been a large one, even with the early cases thrown in. In the future the results will be more favourable. All the deaths at St. George's occurred in the first 200 cases. We have now had more than 320 operations without a single death. During the period that any operation is establishing itself in the surgical world, the operators are of necessity less experienced and handy in its performance than they subsequently become, and are less able to differentiate between suitable and unsuitable cases. It is very difficult to determine the risks run by a person with a reducible hernia. Amongst the educated and leisured class wearing a truss, the risks must be very small, but amongst the ignorant and hard working they are undoubtably appreciable. How often amongst the latter class does one see either an ill-fitting truss, a truss improperly applied, or even put on over a hernia, part of which has perhaps become irreducible. The risks of strangulation under such circumstances become very real, and strangulation, even in a large city within hail of surgeons accustomed to deal with such cases, is a serious matter.

When there is less dread of operation, less dread of hospitals amongst the poor, no doubt such strangulated cases will be operated on even earlier than they are now. Mr. Jones has helped me to look up the cases of strangulated hernia submitted to operation during the last ten years at St. George's Hospital, and they are as follows:

	Inguinal.	Femoral.	Umbilical.
1892 .	10 (3 died)	10 (3 died)	2
1893 .	10 (2 ")	5 (1 ")	1 (1 died)
1894 .	8 (2 ")	13 (3 ")	—
1895 .	11 (3 ")	9 (2 ")	—
1896 .	5 (1 ")	12 (2 ")	—
1897 .	12 (1 ")	13 (1 ")	7 (4 ")
1898 .	13	6	1 (1 ")
1899 .	4 (1 ")	9 (1 ")	2 (2 ")
1900 .	14 (1 ")	8 (1 ")	2
1901 .	16 (3 ")	10 (2 ")	1
	103 (17 died)	95 (16 died)	16 (8 died)
	214 cases, 41 deaths.		

I should like, before concluding, to consider for a moment the causes of relapse after operations for radical cure, and amongst these any want of asepsis at the time of operation must be prominent. If the wound fails to heal by first intention, if there

is suppuration, the likelihood of important buried stitches coming away is much increased. Again, the wound may heal promptly, and yet, owing to some defect in the material of the sutures used, these may subsequently work their way out. If a patient suffer from cough,—as in chronic bronchitis—if he is the subject of anything that causes straining, *e. g.* stricture of the urethra, his chances of relapse soon after the operation are increased. I have met with instances, not, however, in my own practice, of relapse under such conditions. The wearing of a belt round the waist would seem to throw greater strain upon the lower abdomen and region of the rings. I have met with cases where gymnastics, as practised in the Army, under these conditions have seemed to me to have caused hernia. Re-rupture should not be called relapse. No guarantee can be given that a person operated upon will not, if the provocation be excessive, be liable to rupture. It is possible for healthy men to rupture themselves—much more possible for those who have been born with a hernial tendency.

The efficiency of the operation performed, and the efficiency of the operator are also important factors. Every one who has operated much for hernia must have greater confidence as to his results after such experience than when he was doing his early cases. Surgeons are at present divided in their opinion as to what constitutes an efficient operation, but I believe the time will come when there will be agreement amongst them that obliteration of the inguinal canal is a *sine qua non* of such efficiency. Too early resumption of heavy work and insufficient rest in the recumbent position after the operation must also be mentioned as predisposing to relapse.

Assume that the mortality after operation for radical cure is 1 per cent. and that relapses, so-called, are 5 per cent.—and I do not believe they will in the future be nearly as high as this—are we right in recommending the operation? Undoubtedly to those who have to lead an active life, and, personally, I would go further than this, and unless there is some special reason to the contrary, I would recommend it to all otherwise healthy, young, and middle-aged people. In the vast majority of cases union after operation is by first intention and the cure radical, and allowing that a small percentage relapse and re-rupture themselves, are they much the worse than they were before? They have failed to escape truss life and its dangers, but that was their lot before operation. I usually let my patients be up and about again in three weeks, but if children I keep them at rest for at least six weeks, chiefly as a guarantee that they do not over-exert themselves too soon. It is needless to say that for some months after patients begin to get about again they should avoid excessive activity or strain.

CLINICAL REMARKS ON SOME CASES IN WHICH STRICTURE OF THE URETHRA HAS BEEN PERMANENTLY CURED.

By C. MANSELL MOULLIN, F.R.C.S.,

Surgeon to, and Lecturer on Surgery, London Hospital.

I HAVE chosen this subject for my remarks to-day to some extent because we have recently had a number of interesting cases of stricture in the wards, but chiefly because some of them throw a good deal of doubt upon the generally received doctrine that stricture of the urethra is incurable, and will inevitably return. In fact, they do more than throw doubt upon it, they prove that such an absolute statement is quite unjustifiable, and I have no hesitation in saying that it ought not to be made. A large proportion of cases of stricture of the urethra can be cured, and cured permanently, if suitable measures are adopted. Of course I am only speaking of cases of organic stricture. Congestion and muscular spasm are always present as complications in every case of stricture, increasing the difficulty of dealing with it, and making all the symptoms worse. But though they may of themselves, without the assistance of any structural narrowing, close the urethra so completely that no urine can be expelled, the obstruction they cause is not a stricture, and cannot be called one.

Two of these cases had been under treatment in our wards just ten years ago, and had had nothing done for them since. No catheter had been passed for them, and they had not passed one for themselves, though they had been told to do so, and had been given a catheter for the purpose. Internal urethrotomy had been performed in both, and a No. 11 English soft catheter passed in each case without the slightest difficulty. Ten years, of course, is not a permanency, but I think you will agree with me that if no recontraction takes place in that space of time, without anything having been done to prevent it, the chance of its occurring later is decidedly not great.

Some while ago we had in the wards two other cases in which, after all sorts of other measures had been tried and had failed, a cure had been effected by external urethrotomy. In one of these twenty-six years had elapsed since the operation had been performed, and in the other eighteen. No instru-

ment of any kind had been passed in the meanwhile, and there was no trace of recontraction. A No. 12 English entered the bladder without difficulty, and without even giving the sensation of passing over a rough surface. Several similar cases have been recorded by Syme. In one, of which he gives a full account, dilatation had been practised on several occasions; internal urethrotomy had been performed twice, and various other measures had been tried without obtaining more than a transient degree of relief. External urethrotomy was performed, and thirteen years after there was no sign of recontraction, although in the meanwhile none of the usual steps had been taken to prevent it. In another, in which there was a post-mortem examination two years after the operation, it was found not only that the urethra was not diminished in diameter at the site of the old stricture, but it was absolutely widened. There was a funnel-shaped dilatation on the floor, as if the cicatrix had yielded under the pressure of the urine.

Bickersteth has recorded another equally striking. In 1867 Syme's operation was performed in a case of traumatic stricture with urinary fistula of the worst type. In 1869 the man was known to be quite well, although nothing had been done to prevent recontraction. After his death in 1885 the urethra was removed and examined, and, as in the case of Syme, which I have already mentioned to you, the urethra was dilated rather than contracted at the seat of the old stricture. I could tell you of other cases, too, some under my own care, others published in the journals, but these, I think, are sufficient to convince you that such a thing as permanent cure of stricture of the urethra is possible, even in the very worst cases, provided—and this is the point to which I wish particularly to draw your attention—the means adopted are suited to the end in view. Internal urethrotomy succeeds in some. External urethrotomy succeeds in others, and it will succeed, as in one of my own cases, and in another, which I have quoted to you on Syme's authority, even after internal urethrotomy has been tried and has failed. Dilatation, too, I have no doubt, will succeed in comparatively recent cases, though I have no hospital statistics to prove it to you. Why is it, gentlemen, that sometimes one method succeeds, sometimes the other? Why is it they are all of them credited

with such frequent failure? and, given a case of stricture, how are we to tell beforehand which method of treatment we ought to employ, and what degree of success we may expect?

Well, gentlemen, certain cases may be put on one side at once. I do not say that all cases of stricture of the urethra are curable. Some, I believe, are not, except perhaps by excision. They are not of necessity the oldest cases, or those which have been longest neglected. Nor are they necessarily the narrowest ones, or those in which there is the greatest amount of perineal induration. In one of my most successful cases I had to cut through nearly two inches of dense inflammatory exudation before I could reach the urethra from the perineum. The incurable cases are those in which the whole thickness of the mucous membrane of the urethra has been destroyed at some one spot by ulceration. This, of course, leaves a scar extending into the submucous layer, and perhaps deeper still, and you know the physical properties of such scars. They contract, and they always will contract, in spite of all you do. Such cases are really, I believe, incurable except by excision; but fortunately, lest you might be induced to promise your patient more than you can perform, there are several features about them by which you can nearly always tell them beforehand. There have been several in our wards of late. One, you may remember, was a middle-aged man in whom there was a dense scar at the meatus, due to an old syphilitic chancre. This had been incised several times already, and, like all such scars, had contracted again in spite of occasional attempts at dilatation. If it is neglected it will inevitably continue to contract. In another, a young man, only twenty-four years of age, there was a history of rupture of the urethra in days gone by. The ends had not been sutured together. Suppuration had followed, and as always happens, had given rise to the production of the most obstinate form of stricture known. It had been dilated several times already, with temporary benefit, and all that we could do was to dilate it again, as for some unknown reason of his own the patient would not allow us to put into practice the only method of treatment that is of any real use in such cases—excision of the whole stricture and suture of the urethra across the gap that is left. In a third, a patient who was suffering from extravasation of

urine, there was a history of an impacted calculus. In a fourth the stricture was probably caused by the injudicious employment of a catheter during an acute attack of gonorrhœa, for the symptoms began to set in within six months, a much shorter time than usual, and one that points to some exceptional state of things. In all of these there had been destruction of the whole thickness of the mucous membrane at some spot by ulceration; and, as you know, such destruction does not admit of restoration. The injury is permanent.

Cases of this kind do not, however, form the majority, or, I believe, even a large proportion of the cases of stricture that we meet. In the greater number there is no such ulceration, and therefore no such cicatrix. There may be, and often is, an indurated mass extending into the corpus spongiosum, so that it is easy to feel the stricture from the perineum. But the mucous membrane, though it may be so altered in texture as to be scarcely recognisable, is not destroyed. If the stricture is slit up the lining membrane can be dissected off from the deeper strata and spread out to nearly its full width. This makes an absolute difference. There is an absolute difference between the contraction of a scar following ulceration and the narrowing of the urethra owing to the deposit in its walls of an exudation caused by persistent inflammation. The one cannot be absorbed, the other can, and the problem before us is to devise some means for putting a stop to this persistent inflammation and for causing the absorption of its products.

The solution is not so difficult if you think for an instant of the reason why this inflammation is so persistent. The gonococcus is not the cause. It is, of course, primarily responsible for the stricture, but it is not the cause which keeps up the irritation and makes the stricture grow worse and worse. The cause that does this is the stricture itself, which is at one and the same time the consequence of the original inflammation and the reason for its obstinate persistence. A stricture keeps on working in a vicious circle. Itself the result of the organisation of lymph in the walls of the urethra, it acts as a constant irritant, causing more and more lymph to be poured out; and this, in its turn, as it becomes organised, narrows the stricture still more and makes the irritation worse. With such a condition of things it does not require

much imagination to picture the result, and, what is much more important, how that result is to be prevented. If the cause of the stricture constantly growing worse is the stricture itself, the only way to effect a permanent cure is to remove the stricture thoroughly, once for all; and if the stricture is thoroughly removed, and no other source of irritation allowed to remain, there is absolutely no reason why the inflammation should persist, or the stricture return.

I know that such a statement as this sounds like rank heresy. The doctrine you always hear is that stricture is incurable. Once a stricture always a stricture, and I quite admit that as stricture is usually treated this is perfectly true. Recontraction always does take place. The patient always has to pass a bougie for himself afterwards. But this only happens because sufficient pains are not taken to remove the cause thoroughly. If the stricture is treated effectually, and the urethra freed from all source of irritation for a sufficient length of time, the lymph that has been organised will undergo degeneration and be absorbed, and the tissues will regain their natural flexibility. Permanent cure is quite possible in a large proportion of cases, and in some of the very worst; and the instances I have brought before you are sufficient to prove it.

Well, gentlemen, how is this to be done? How is the stricture to be removed and the urethra given perfect rest for a sufficient length of time? There are, roughly speaking, three methods at our disposal for the treatment of stricture: dilatation and internal and external urethrotomy. These methods, of course, vary greatly in severity, and are not to be employed indiscriminately. Opinions differ greatly as to their respective merits, but with regard to one of them I have no hesitation in telling you that if a stricture can be felt distinctly from the outside you must divide it from the outside if you wish to cure your patient. External urethrotomy is unpopular. There is an open wound, the urine escapes through it, the patient is confined to bed, and he is longer getting well than after any other method of treatment—quite sufficient to condemn it in the patient's eyes; but these are the very reasons why it succeeds, and succeeds in the worst cases, in which other methods have been tried and have failed. External urethrotomy divides the stricture far more

thoroughly, and gives the urethra far more perfect rest, for a greater length of time, than any other method. Before the wound has closed, and the urine has resumed its natural course, there has been time for all the inflammatory exudation to disappear, and no source of irritation is left behind.

Dilatation cannot do this in these cases. In fact, many of these advanced strictures cannot be dilated. Nor is internal urethrotomy much better, for the division of such a stricture from the urethral surface is not sufficiently thorough unless the incision is carried through almost to the skin, a proceeding which I should advise you not to adopt.

If, on the other hand, the stricture cannot be felt from the outside, if it seems probable that the corpus spongiosum is not involved, and that the exudation is limited to the mucous and submucous layers, it may be treated from the urethral surface, either by dilatation or division. Personally, as you know, I prefer division for all but quite recent cases, as it is much more speedy, involves much less instrumentation, is more efficient, and is absolutely free from the risk of rigors and septic absorption if properly carried out; but whichever of the two you adopt, the essential point to remember is that the *calibre of the urethra at the strictured spot must be enlarged to the greatest width of which it was capable before the inflammation set in*. If you do not do this, and do it thoroughly, you leave what is potentially a stricture behind, and fail to cure your patient. There is no definite relation between the size of the penis and that of the urethra. You cannot ascertain the width to which any part of the urethra is capable of being distended by external measurement. The only way is to ascertain in each case the extreme width to which the normal urethra in the immediate neighbourhood of the stricture can be stretched, and then to dilate the strictured part until it is at least of the same width, whether this is 20, or 22, or even 24 of the English scale.

If you do not do this, you leave in the urethra a part which, if not actually smaller than the rest, is certainly not capable of an equal degree of distension. It is a potential stricture, and it will contract again. It is not fair to say that the stricture has returned, and that it is the fault of the patient for not passing bougies sufficiently often. The stricture has not been removed, and the treatment was inefficient.

A CLINICAL LECTURE ON MITRAL REGURGITATION.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, November 20th, 1901.

By P. HORTON-SMITH, M.D., F.R.C.P.,
Assistant Physician to the Hospital.

GENTLEMEN,—In the last lecture Dr. Maguire brought before you the subject of aortic regurgitation. To-day I propose to deal with the similar lesion as it affects the mitral orifice.

Relative frequency of mitral regurgitation.—Let us consider first of all the relative frequency of the lesion when contrasted with other varieties of morbus cordis. On this point there has been some difference of opinion, and with the view of adding to our knowledge upon the subject I have analysed the cases of heart disease which have so far been under my care at the Brompton Hospital. These number exactly 100, and the various forms of morbus cordis met with among them may be seen from the following table:

TABLE I.—*Showing the result of an analysis of 100 consecutive cases of morbus cordis attending the Brompton Hospital.*

Mitral regurgitation	37
Mitral stenosis	18
Double mitral disease	16
Aortic regurgitation	8
Aortic and mitral regurgitation . .	5
Aortic regurgitation and double mitral disease	5
Aortic regurgitation and mitral stenosis	4
Aortic regurgitation and stenosis .	1
Aortic regurgitation and stenosis with mitral regurgitation . . .	1
Aortic regurgitation and stenosis with mitral stenosis	1
Aortic stenosis and mitral regurgitation	1
Aortic stenosis and double mitral disease	2
Aortic and mitral stenosis	1

From these figures it is clear that in this series mitral regurgitation was the commonest of the four valvular diseases; next comes mitral stenosis, then aortic regurgitation, and lastly, and least common

of all, aortic stenosis. This predominance of mitral regurgitation is also borne out by the in-patient records of St. Bartholomew's Hospital (see Table II), where, during five years, simple mitral regurgitation occurred in 200 cases, aortic regurgitation in 117, mitral stenosis in seventy-one, while simple aortic stenosis was diagnosed on six occasions only. There can be little doubt, therefore, from these facts that mitral regurgitation is the commonest of all the valvular lesions.

Sex.—In some forms of morbus cordis sex exercises remarkable influence. Thus aortic regurgitation occurs far more frequently in the male, while mitral stenosis, on the other hand, occurs more commonly in the female. With mitral regurgitation, however, this is not the case, and the sexes are affected equally. These facts are well shown in the following table (Table II), where the sex-incidence of the various lesions is given, as deduced from the records of in-patients at St. Bartholomew's Hospital during the five years 1896—1900. The figures are based upon the admirable statistics prepared yearly by the Medical Registrars, Dr. Garrod and Dr. Calvert:

TABLE II.—*Showing the sex-incidence of the various forms of morbus cordis, as deduced from the records of in-patients at St. Bartholomew's Hospital during the five years 1896—1900.*

	Total number of cases.	Male.	Female.
1. Mitral regurgitation .	200	96 (48 %)	104 (52 %)
2. Double mitral disease .	151	60 (40 %)	91 (60 %)
3. Mitral stenosis .	71	30 (42 %)	41 (58 %)
4. Aortic regurgitation* .	117	97 (83 %)	20 (17 %)
5. Aortic obstruction .	6	4	2
6. Congenital morbus cordis	24	8	16

From the above it is seen that while in aortic regurgitation 83 per cent. of the cases were males, and in mitral stenosis 58 per cent. were females, no appreciable difference in this respect could be made out in the case of mitral regurgitation. Thus out of 200 cases 96 were males and 104 females. Sex, therefore, in this instance exercises but little influence.

Ætiology.—As regards their cause, cases of mitral regurgitation may be divided into two great groups:

* Doubtless in a certain proportion of these cases the regurgitation was accompanied by some stenosis of the aortic orifice, but in what proportion exactly this occurred it is impossible to say.

(1) Those in which the disease is due to a lesion of the valve, and (2) those in which the disease results from failure of the muscular tissue surrounding the orifice, the valves themselves remaining natural. To these may possibly be added a third group of cases, in which the regurgitation is due to a disturbance of the nervous mechanism of the heart, but this cause is a much less common one. Let us now consider each of these groups in turn.

GROUP I.—*Those in which the disease results from a lesion of the valve.*—Of the causes which produce such a lesion, *rheumatic fever*, as you are well aware, stands pre-eminent. Thus of 118 cases of mitral regurgitation, dating from an early age, analysed by Dr. Sansom,* seventy-eight gave a history of rheumatic fever, while in thirteen others also it was probable that the primary endocarditis was really rheumatic in nature; that is to say, in certainly two thirds of the cases, and probably in three quarters, the valve lesion was the direct result of rheumatic fever. Although, however, this disease is by far the commonest cause of endocarditis affecting the mitral valve, yet other fevers also may give rise to it. Of these *scarlet fever* is perhaps the best known, eight of Dr. Sansom's 118 cases being directly attributed to this disease. Of the cases which I am bringing before you to-day, one of them is an example of this mode of origin. The patient is a little girl, æt. 7 years, who was quite healthy until she contracted scarlet fever three years ago. Since then she has suffered from cardiac pain and palpitation, and, as you will see, presents the physical signs of mitral regurgitation. At the time when she had the scarlet fever, Dr. Bruce, of the Western Fever Hospital, under whose care she was, wrote to her mother (so she informs me) saying that her daughter's heart had become affected as a result of her disease. There can be little doubt, therefore, that in this case the morbus cordis should be directly attributed to the specific fever, the organism of the disease (possibly the streptococcus conglomeratus of Gordon) having found a lodgment on the valve and given rise to endocarditis, much in the same way as we believe to occur in the case of the more ordinary virus of rheumatic fever. Another disease, again, which may produce

* Article on "Diseases of the Mitral Valve," Clifford Allbutt's 'System of Medicine,' vol. v, p. 984.

endocarditis, leading to chronic valvular disease, is *measles*. Of Dr. Sansom's cases, six were attributable to this disease. Personally, however, I have not as yet seen a case which I could directly trace to this complaint. Of the causes, then, which give rise to endocarditis affecting the mitral valve, and, secondarily, to mitral regurgitation, rheumatic fever is by far the commonest. At a great distance after this comes scarlet fever, and, more rarely still, morbilli. Occasionally, however, other fevers, such as pneumonia, variola, and typhoid fever may produce this disease; but in such cases the endocarditis is nearly always of the malignant type, and the patient usually dies before the disease has time to assume the chronic form. As causes of chronic valvular disease, therefore, they need hardly be considered.

Rupture of the valve.—Mitral regurgitation, however, even when due to disease of the valve structures, may arise in another way to that which we have been considering. Thus it may occur suddenly, as the result of some great strain, from rupture of certain of the chordæ tendineæ, though such an event is infinitely rare. The condition was first described by Corvisart* in 1811, and since then a few similar cases have been recorded. I cannot to-day bring before you any patient who is suffering from such a lesion, but the specimen which I now show you demonstrates very well the pathological appearances seen in such a case. As you will notice, the mitral orifice is greatly dilated (it admitted four fingers on the post-mortem table), and, in addition, several chordæ tendineæ, which passed from one of the large papillary muscles to the adjacent edges of the two cusps, are ruptured. As a result the valves flapped back into the auricle at each beat of the heart, and regurgitation of blood was permitted. Apart from the rupture of the chordæ, the valves themselves seem natural. This specimen was taken from a labourer, æt. 33 years, who had not suffered from any symptoms referable to his heart until he strained himself nine weeks before his admission to the hospital, when putting up a crane. At this moment he was seized with pain over the heart and shortness of breath, which symptoms continued until he came to the hospital. He was then seen by Dr. Habershon (who has kindly furnished me with

the details of the case), and was found to be suffering from mitral and tricuspid regurgitation, and to possess a markedly pulsating liver. He was admitted, and, in spite of treatment, died a month later from syncope, three months from the commencement of his symptoms. From a consideration of all the circumstances of the case there can be little doubt that in this instance the rupture of the chordæ tendineæ resulted directly from the strain experienced three months before death, even though, as the post-mortem appearances seemed to show, the valve itself was previously healthy.

Although, however, I cannot show you a patient suffering from rupture of his mitral valve, yet, to demonstrate the condition, I am bringing before you a man who presents all the appearances of *rupture of an aortic valve*.

The patient is forty-seven years of age, and is a porter in the meat market. According to his history he was quite well in every way until six months ago, never having complained of cardiac pain or shortness of breath, and never having had rheumatic fever. Six months ago, however, when carrying on his back, in the course of his work, half an ox (weighing twenty-two stone), and when making the final effort requisite to lift this great weight from his back on to the cart which was to carry it from the market, he suddenly experienced intense pain in the chest and shortness of breath. After a time he was able to go on with his work and finish it for the day, but since that time he has never been able to do an hour's work owing to the fact that his breath is so short and that he is liable very frequently to severe attacks of agonising pain which shoot down from the heart to the tips of the middle and ring fingers of both hands. On examining his chest you will find that he presents the physical signs of aortic regurgitation. The apex-beat is in the sixth space, half an inch outside the nipple line, while a diastolic murmur is audible at the second right interspace and also at the apex. The second sound at the aortic base is inaudible. The history, then, which in this case is exceptionally clear, suggests strongly that at the moment of the great strain six months ago, a rupture of one of his aortic cusps took place, leading directly to the subsequent regurgitation. I should add that the patient had syphilis twenty years ago, and it is possible that his aortic valves were slightly sclerosed at the time of rupture, the

* 'Essai sur les Maladies du Cœur,' par J. N. Corvisart, Paris, 1811, p. 263.

majority of observers believing that some sclerosis nearly always precedes rupture when this rare condition does occur. That he had not suffered from aortic regurgitation before seems, however, proved by the sudden onset of his symptoms six months ago.

GROUP 2.—*Those cases in which the regurgitation is due not to disease of the valve, but to failure of the muscular tissue surrounding the mitral orifice.*—Regurgitation so produced is not uncommonly combined with great dilation of the left ventricle and of the mitral orifice, the enfeebled muscular tissue of the ventricle yielding and dilating until the mitral cusps can no longer meet and close the aperture. This form of the disease is well seen in the later stages of aortic regurgitation.

In another class of cases, however, no dilation of the orifice is found post mortem, and the regurgitation results simply from enfeeblement and loss of due contractile power of the cardiac muscle. For in this connection it must be remembered that at each beat of the heart the mitral orifice is normally diminished to about half its size (as Ludwig and Hesse have shown), and if this contraction fails to occur mitral regurgitation inevitably results. It is probable that in certain cases of anæmia, of fibroid and syphilitic disease of the heart, or where the nutrition of the muscle is impaired, as the result of disease of the coronary arteries, mitral regurgitation is produced in the manner thus suggested.

GROUP 3.—*Those cases in which the disease possibly arises as a result of disturbance of the nervous mechanism of the heart.*—This class of case has been drawn attention to by Dr. Sansom, who points out that in all probability of late years we have devoted too little attention to the nerves of the heart as a possible source of disease. This observation no doubt is a true one, for when we remember the number and size of the cardiac nerves it is certain that they must perform some definite function in controlling the action of the organ, even though its contractile power rests, as we know now to be the case, in the muscular tissue itself. Theoretically, then, mitral regurgitation might result from defective action of the cardiac nerves, though clinically this cause does not seem to be a common one. It is possible that it is best exemplified in those cases of temporary mitral regurgitation which sometimes complicate

cases of exophthalmic goitre, and a good example of which is given by Dr. Sansom in his admirable article on "Mitral Disease," already referred to. The nervous features of Graves' disease are clearly marked, and it may well be that the cardiac dilation and consequent mitral regurgitation which sometimes supervene during the disease, are really the result of defective innervation. Of such cases, however, I have as yet seen myself only one possible example.

Morbid anatomy.—On this subject I may be brief. As you are well aware, when the valves in a case of mitral regurgitation are diseased, they are generally found on the post-mortem table to be shrunken and thickened, while the chordæ tendineæ also are very often shortened and contracted. Not uncommonly too the adjacent edges of the valves may be found united, whence a certain amount of stenosis may also result. These changes in the valves, however, are not the only pathological appearances found, for in nearly all cases of mitral regurgitation certain secondary changes in the cavities of the heart also occur, their extent varying with the severity of the lesion. Thus the left auricle will, in general, be found dilated and somewhat hypertrophied, owing to the reflux of blood which takes place into it from the ventricle. As a consequence of this, and of the difficulty which the auricle finds in emptying itself, the tension of the blood in the pulmonary system is increased. More work is thus thrown on the right ventricle, which consequently shares also in the dilatation and hypertrophy. As a result, also, of these combined changes, more blood is thrown during diastole into the left ventricle, and as a rule this, too, shows in some degree alterations similar to those met with in the preceding cavities, though in general in the left ventricle these changes are much less marked. In consequence of the alterations thus rapidly sketched out, the heart increases considerably in size, and may come to weigh as much as twenty ounces.

Symptoms.—Let us now pass to the symptoms of the disease. These depend to a great extent on the severity of the lesion and on the measure of compensation which has been acquired. If the lesion is slight and compensation good, there may be no symptoms at all. In general, however, the patient is not so fortunate, and for one cause or another is driven to seek advice. What, then, are

the symptoms of which he now complains? To answer this question I have analysed the histories of thirty-two of my cases, and the result is given in the following table :

TABLE III.—*Showing the symptoms for which the patient sought advice.*

	No. of Cases.
Cough, or	18
Cough and shortness of breath	
Palpitation	7
Cardiac pain	
Oppression over heart	
Faintings	2
Hæmoptysis	1
Debility and	2
Loss of appetite	
Dyspepsia	2
Total number of cases	32

From these figures, which agree well with the experience of others, it will be seen that *cough, shortness of breath, palpitation, and cardiac pain* are the chief complaints which cause the patient to consult a doctor. Later on, as we know well, these symptoms become more marked ; dyspnoea becomes extreme and orthopnoea results ; sleep becomes broken, and in the later stages dropsy also is a marked feature.

Appearance.—Let us now consider for a moment the appearance which a patient with mitral regurgitation presents when he first comes under observation. In seventeen of my cases I made a careful note on this point, and the following is the result obtained :

TABLE IV.—*Showing the general appearance presented by the patient when first seen.*

Appearance.	No. of Cases.
"Very good"	1
"Very fair"	5
Poor and anæmic	10
Marked anæmia and emaciation, suggesting phthisis	1
Total number of cases	17

It may be added also that in no case was definite cyanosis observed. The deduction, then, which may be drawn from these facts is that *in the majority of early cases of mitral regurgitation*

the appearance presents nothing special. The patient, as a rule, looks somewhat anæmic, but nothing more definite can be stated. It is only in the later stages of the disease that the face becomes cyanosed and that oedema occurs. The appearance, therefore, can but rarely help us in forming a diagnosis in an early stage of the disease.

(To be concluded.)

Vomiting during Recovery from Anæsthesia.—With chloroform for the anæsthetic, vomiting occurs in fewer cases, but when it does it is later in the stage of recovery, often when the patient is quite conscious. It is then very annoying, and will be distinctly remembered by the patient. When vomiting occurs after chloroform it is sometimes very severe, being both very frequent and accompanied by severe straining, and in some patients by a marked tendency to syncope. For the slight cases no treatment beyond abstinence from all food for three or four hours is necessary. Small pieces of ice are frequently given to be sucked, but a teaspoonful of water as hot as the patient can take it, given at intervals of about a quarter of an hour, will probably be of more service in checking the vomiting. With this hot water a minim of the tincture of nux vomica may be given. If the water can be taken really hot, it is best given in a china spoon, as a metal one may burn the lips. Should the vomiting not yield to these remedies, from ten to fifteen grains of bicarbonate of soda may be given in a cup of hot tea or coffee. Should these fail, the patient must be treated on the same lines as one suffering from persistent vomiting from some other disease. Ice-bags and a mustard leaf to the epigastrium have been recommended, and occasionally the stomach has been washed out with a good result.

The above is taken from a book just published by Longmans, Green, and Co., entitled 'A Practical Guide to the Administration of Anæsthetics,' by Dr. Probyn-Williams, the senior anæsthetist at the London Hospital. Within the short compass of 200 pages the author has succeeded in supplying all the essential points in the practical administration of anæsthetics. The excellence of this work will undoubtedly secure for it a very wide field of usefulness.

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A CLINICAL LECTURE

ON

PARAPLEGIA.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London,
November 16th, 1901.

By J. S. RISIEN RUSSELL, M.D., F.R.C.P.,
Physician to Out-patients at the Hospital, and Assistant
Physician to University College Hospital.

GENTLEMEN,—The subject which we have chosen for to-day's lecture is paraplegia. By paraplegia we mean an affection in which the lower limbs are paralysed. Commonly the lower part of the trunk is also involved. You sometimes hear "cervical paraplegia" spoken of. By that is meant a paralysis affecting the two upper limbs as opposed to the lower. When you hear the term paraplegia applied without any qualification, what is meant is a bilateral paralysis involving the lower extremities; and as I have said, commonly you have, in conjunction with the paralysis of the lower extremities, involvement of the lower part of the trunk in cases where the lesion is in the spinal cord. Paraplegia may result from lesions in different parts of the nervous system. It most commonly results in cases of spinal cord affection. On the other hand, you may have the peripheral nerves affected, and paraplegia resulting in consequence. Again, an affection of the brain, instead of causing hemiplegia, as is most common, may give rise to paraplegia. So that some lesion involving both cerebral hemispheres may give rise to paralytic manifestations in the lower limbs practically alone, the paralysis then being spoken of as paraplegia. There are a great many spinal cord affections which lead to paraplegia, so that not only is paraplegia due to lesions in the different parts of the nervous system, but, taking one part, as, for instance, the spinal cord, you find a large number of different lesions leading to this condition. The first case which I bring

before you this afternoon is one of spastic paraplegia. What we find in this man is that there is a disability in connection with his lower limbs, that disability being caused by spasticity in conjunction with motor weakness. I will let the man attempt to walk, so that you may see his gait, but before doing so let me refer to certain other points in connection with the case. This condition from which he is suffering is one in which the motor elements of the spinal cord are alone involved. The disability is due to some lesion of his upper motor neurons, causing a weakness with spasticity. Similar motor disability might result from implication of his lower motor neurons, that is to say, the anterior horn-cells in his spinal cord. But under such circumstances you would not expect spastic phenomena in conjunction with the motor weakness; you would expect to find the flaccid form of paraplegia, and no evidence of spasticity. The fact, then, that this man has a spastic condition of limbs points to involvement of the upper motor neurons, that is to say he has some lesion of the lateral columns of his spinal cord, giving rise to the spastic condition which we find in his lower extremities. As regards his disability, it is purely motor. We find no evidence in him of any blunting of sensibility. I will test him before you in order that you may see whether he can appreciate tactile impressions. As the man has been in the hospital you may take it that he has been carefully tested, and I can assure you there is no evidence of blunting of cutaneous sensibility. Next let us look at the condition of his tendon-jerks. We find that he has marked exaggeration of the knee-jerk on both sides, which is in accordance with what you would expect in view of the fact that I told you we were dealing with a case of spastic paraplegia. Next we test for ankle-clonus. At the present time he has no evidence of ankle-clonus. We might have found, in conjunction with the increased knee-jerks, ankle-clonus on both sides. We next examine the state of his plantar reflex. On stimulating the sole of a normal individual's foot you expect the toes to flex. On stimulation of this man's foot, however, you find that the great toe extends. You will see the same phenomenon on the other side, namely, a drawing up of the great toe, known as "Babinski's sign." This indicates an affection of the pyramidal system, and supports

the diagnosis I have already mentioned. I will now ask him to walk, so that you may see his mode of progression. I think it is abundantly evident that there is marked disability in connection with his lower limbs, that he has spasticity associated with motor weakness. He cannot clear his feet from the blanket on which he is walking, and he has to raise his pelvis with each step in order to get the foot off the ground. There his disability ends, for when we come to examine his upper limbs we find he has good power in them, and that he can execute all movements of the upper limbs perfectly. He has no inco-ordination of the upper limbs, and there is no evidence of any muscular atrophy of his hands. You may take it from me that the same applies to other parts of the limbs. So, too, the cranial nerves are all intact; we can find no evidence of any defect in the distribution of any of them. When he replies to my question as to how long he has been ill you will notice that his speech is natural. So that we have here a man presenting merely the features of spastic paraplegia, namely, rigidity in connection with motor weakness in the lower limbs, with exaggeration of his tendon-jerks, and with the peculiar alteration of the plantar reflex which we expect in affections of the pyramidal system, but without any sensory defect. Now, gentlemen, in the examination of such a case never omit to look at the back. In any case of paraplegia it is your duty to examine the back of the patient. I will not strip this man now to show you his back, because he presents nothing peculiar for you to see; there is no abnormality about his spinal column. But in such cases you may find some deformity or curvature which gives you the clue as to the cause of the paraplegia. So, too, in the absence of deformity, you may find localised tenderness at some spot in the back indicating the seat of the lesion. This man has slight diffuse tenderness in the lower thoracic and lumbar regions, but nothing localised; it is a perfectly superficial general tenderness.

What, then, is the nature of the lesion in such a case? We regard it as a primary lateral sclerosis. The history of the case supports this view, just as the manifestations you see at present are in harmony with it. The history of the man is as follows:—He is forty-one years of age, and is said to have had an attack of "rheumatism" about a year ago, in which

he says that his joints were swollen, especially the knees and ankles. He had had similar attacks of rheumatism before on several occasions during the last eleven years, but after the last attack he gradually developed stiffness and weakness in his lower limbs. The only other phenomenon which he noticed was in connection with his right hand. He says there was a certain amount of stiffness and numbness in his right hand, so that he had a difficulty in buttoning and unbuttoning his clothes. However, objectively nothing has been found in connection with that arm, as regards any motor or any sensory defect, and there is no disturbance of co-ordination. We see, then, that as far as objective signs are concerned his disability is limited to the lower limbs. It is therefore a case of primary lateral sclerosis. That is the diagnosis to which we are commonly driven clinically. It, however, is not based on any very substantial support from morbid anatomy. I say, as far as the pathological anatomy of the affection is concerned, we have very slender evidence indeed on which to build up a distinct disease. Clinically, however, there is this well-marked type of affection, in which you have a progressive spastic paraplegia of gradual onset, without any defect of sensibility, and in which the condition often shows little tendency to spread beyond the lower limbs. In some cases you may find some spread to the upper limbs, so that there is spasticity of the arms, but the disease does not spread further. There are one or two cases on record in which the lesion found has been a lateral sclerosis of the spinal cord, that is to say, sclerosis of the pyramidal tracts and nothing else. But it is doubtful whether those cases have been sufficiently examined with regard to lesions in the brain. In the vast majority of cases where a primary lateral sclerosis has been diagnosed and where there has been an opportunity of verifying the condition by necropsy, some affection has been found other than a simple primary lateral sclerosis. From the clinical standpoint we find that many of the cases which begin as a primary lateral sclerosis go on later to other affections, the varieties of which I have not time to relate to you. But I would especially mention disseminate sclerosis, when the spastic paraplegia occurs in a young person. Nevertheless, gentlemen, there is undoubtedly a clinical group in which we can find nothing else than we have found in this man, and

in which, hypothetically, we must suppose that we have a sclerosis of the lateral columns of the spinal cord, which is the sole lesion accounting for the motor disability. It has been said that the reason why we have not collected a sufficient number of post-mortem records on which to build up a pathological anatomy for these cases is that we do not follow them to the end; and it is also said that we should look for them in the various infirmaries, for there these cases are likely to terminate, in that they are of slow progress and last for many years. This argument applies to this country, but it does not apply to France, where, for instance in the Salpêtrière, these patients are kept under observation for years and are followed to the end. Yet French observers are in the same difficulty as ourselves in regard to this clinical entity.

The next patient I bring before you is another case in which you will find the manifestations of a spastic paraplegia. I bring him directly after the last patient in order to show you that in so far as the motor phenomena are concerned, they are closely similar to those of the individual you have just seen. This man at the present time presents the features of paraplegia. His disability is in the lower limbs, and he has some spasticity in association with the motor weakness, the spastic condition being chiefly noticeable in his right leg. He has also a certain amount of dropping of the feet, so that he cannot clear the ground perfectly in consequence of the weakness of the extensor muscles. His knee-jerks, as in the case of the other man, are markedly increased, the slightest tap producing the contraction very decidedly. Further, this patient has marked ankle-clonus on both sides. The other patient you saw had no definitely persisting ankle-clonus, only a slight tendency to this phenomenon. We now examine this man's plantar reflex, and we see the same phenomenon that we saw in the other case, namely, the "extensor response," extension instead of flexion of the toes, as would occur in a normal individual. So far, then, the pictures in the two cases are similar, and, further, there is no affection of the upper limbs. On examination of his hands we find no evidence of muscular atrophy. His grasp is good on both sides, and, as in the other case, we find no evidence of any inco-ordination. You may take it from me, further, that his cranial nerves are intact, no abnormality having been

determined in connection with any of them. We have here again, then, a condition of paraplegia in which the disability is limited to the lower limbs. Wherein does this case differ from the last? We have not yet tested the cutaneous sensibility. We tested the last man's, and were able to determine that there was no blunting to tactile impressions; I also told you of the observations which had been made showing that all forms of sensibility were intact. In this individual there is very little remaining of any blunting of sensibility, but when the patient came into the hospital he presented manifestations of anæsthesia such as you see here in the chart I show you. The main point is that he had marked loss of sensibility to touch and to pain on the left leg, chiefly in front. In addition to that he had blunting of all forms of sensibility, extending up to the level of just about the umbilicus, that is to say, the level of the ninth thoracic root-level. Posteriorly the anæsthesia extended up to about the second lumbar spine. The anæsthesia has now cleared up considerably, and I am not sure whether we shall now be able to demonstrate any blunting of sensibility. Let me try. I think it is clear to you that on the limb where he had the marked blunting of sensibility before there is still some defect. We will now see if he experiences any pain when pricked with a pin in that region. He feels the prick of a pin as a touch on the left leg, whereas on the right leg he recognises it to be a prick. Although he can appreciate a painful impression on the right lower limb it is not so distinct as on the upper limbs, and there is distinct evidence of blunting of sensibility to pain on the left leg. Similarly he had blunting to thermal impressions, as shown on the chart, so that he did not accurately appreciate heat and cold impressions. We have, therefore, this marked difference between these two cases: that although from the motor side the manifestations are similar, we have here sensory defects in conjunction with the motor. In short, there is something in the spinal cord which is interrupting the sensory conducting paths as well as the motor conducting paths. By consulting the history of his illness we find what that something is. He is twenty-three years of age. Rather more than three years ago he contracted syphilis, and about two years after he began to experience the symptoms of his present disability. The first thing that he noticed wrong was pain in

the chest and in the back between the shoulders. In association with that he had a certain amount of constriction around his body, just below the level of the umbilicus. At that time he also suffered from retention of urine, and went into a hospital, where he had his urine drawn off. Following on this retention of urine, for which he went into the hospital, he developed incontinence of urine and incontinence of fæces. Therefore, he had loss of control over both bladder and rectum following upon the first manifestations of disability, namely, retention. Now, at any rate for a week after the onset of his illness, there was no marked loss of power in his lower limbs. He is sure of that, but he is not clear as to when the disability in the lower limbs began in that he was kept in bed. All that he can say is that a month later, when he got up, his legs were weak. He could stand, but his limbs shook when he attempted to walk, in addition to which he could not walk straight. At first he appears to have become a little worse, and then he began to improve, and there had been slight improvement up to the time when he came into this hospital. When he was admitted here, in addition to the motor disability in the lower limbs, there was the anæsthesia to which I have alluded and complete loss of control over both his sphincters. There has been improvement since he has been in the hospital, not only as regards the sensory disturbance, but also with regard to motor power. There has also been some improvement in connection with his sphincters, so that he is able to retain his urine longer than was possible when he first came here. What I have told you about the patient justifies the belief that we are dealing with a case of syphilitic myelitis. This patient has to thank the specific disease, contracted somewhat more than three years ago, for a myelitis which is responsible for the interruption of the motor and sensory paths in his spinal cord and for the disturbance of the action of the sphincters.

I now want to show you another man. First let us make sure that we are dealing with a case of paraplegia before we go on to a description of the other features of the case. He can only walk with assistance, and even then he can only do so in the feeble way which you see. In addition to this man's motor weakness there is evidence of spasticity of his lower limbs. There is marked

exaggeration of the knee-jerk on both sides. He has ankle-clonus and tremor of the leg higher up as well ; indeed, there is so much clonus above that it is liable to obscure the ankle-clonus. Next we try to elicit the "extensor response." This we can get on the left side, but his feet are very cold, and at present I am unable to obtain it on the right. There is no sensory disturbance in his legs. So far, then, the case conforms more or less the picture of the first case we saw this afternoon : loss of motor power, spasticity in connection with the motor disability, and no sensory loss. But that the case is not merely one of spastic paraplegia, so-called primary lateral sclerosis, is made evident by a glance at the man's upper limbs, for you can see that we are dealing with something far more widespread than mere spastic paraplegia consequent upon implication of the pyramidal tracts. What we notice in connection with the upper limbs is marked wasting of the hand muscles. There is marked wasting of the first dorsal interossei, the thenar and hypothenar muscles on both sides. If we strip him we find the same thing in connection with the muscles of the forearm. There is excessive wasting on the ulnar side in conjunction with the wasting of the small muscles of the hand, which I have shown you. So we have here spastic paraplegia combined with muscular atrophy of the upper limbs. In short, we have an affection which, in addition to involving the upper motor neurons, has involved the lower motor neurons in the cervical region of the cord, causing the muscular atrophy which you see in this man's hand. Now, can we get any further help in this case? I will ask him to speak, and you will notice that in addition to the slowness there is a thickness and a slight nasal tendency in his utterance. In addition to this we find that on attempting to drink liquids he has difficulty in swallowing, and he gets some regurgitation through the nose. So we have in him some disturbance in connection with his bulb as well as that in the cervical region of his cord and in the pyramidal tracts. He can blow out his cheeks and he can whistle fairly well. On examining his tongue we find slight atrophy, as indicated by two longitudinal furrows on either side. Another thing which we notice about his tongue is that there is some fibrillary tremor. He protrudes it in a stiff manner, and if I tell him to waggle his tongue he does so imperfectly, and he

moves his lower jaw from side to side in the effort. He can, however, push his cheek out with his tongue with fair force. In addition to the points we have already observed he has weakness of his neck muscles ; the flexors are stronger than the extensors. In this case, then, the spastic paraplegia is merely part of a more general condition, the malady being amyotrophic lateral sclerosis, which is an affection in which you have defect of the upper motor neurons, causing the spastic phenomena which we have observed, and of the lower motor neurons, causing the muscular atrophy which we have also seen. Degeneration of the lower motor neurons in the cervical region of the cord has caused atrophy in the upper limbs, and similar defect in the bulbar nuclei has caused the affection of the palate and tongue. As regards the spastic phenomena, I can show you evidence of implication of the pyramidal path higher up than in the part concerned with the lower limbs, in the state of the jaw-jerk. You will notice that when I strike his lower jaw it goes off almost into a state of clonus. That indicates involvement of the upper motor neuron, and is of the same significance as the exaggeration of the knee-jerks and the ankle-clonus which we have seen in connection with his lower limbs. This man, æt. 38 years, attributes his illness to an accident. He was not injured, but he suffered from shock. His disability did not, however, come on for about six months after this shock. With regard to other antecedents he does not appear to have had venereal disease, but he admits to excess in alcohol, and he has been exposed to weather. As regards the mode of onset of his illness, as you have heard him say, he was jumping into an omnibus when he first noticed the weakness of his right leg. Four or five months later he noticed that his left lower limb became similarly affected, and then his left hand became weak, and more recently the right hand has become similarly affected. Much more recently he has begun to develop the difficulty with regard to his speech and difficulty in connection with swallowing. The whole history of his illness extends a little over two years.

The next patient, a man æt. 40, has also spastic phenomena in connection with his lower limbs. He has an exaggerated knee-jerk on both sides, more so on the right than on the left. He also has ankle-clonus on the right side, but not on the

left. With regard to the plantar reflex, the "extensor response" is present on the right side, but I am unable to obtain it on the left. As a matter of fact, the "extensor response" has been obtained on both sides. On looking at his upper limbs we again find that, as in the last case, the spastic phenomena below are only part of the picture. Here we have another individual presenting muscular atrophy. The muscular atrophy is marked in the small muscles of the right hand, that is to say, the interossei, the thenar and hypothenar muscles, and he has well-marked claw-hand. On the left side there is only slight atrophy; there is nothing pronounced as in the case of the other hand. There is especially some wasting in the first dorsal interosseous space, but apart from that there is but little defect in connection with this hand. Is this case similar to the one we have just seen? Are we again dealing with a case of amyotrophic lateral sclerosis? There are spastic phenomena in the lower limbs and an atrophic condition of the upper limbs consequent upon involvement of the lower motor neurons in the cervical region of the cord. Let us see if he has anything which will help us further in respect to other phenomena presented by the other case. He has no evidence of any bulbar affection, but it is not necessary that you should have bulbar manifestations in order to establish a diagnosis of amyotrophic lateral sclerosis. All that this man has would suffice for the diagnosis of amyotrophic lateral sclerosis, without its being necessary to demonstrate any bulbar manifestations. Is this case, then, one of amyotrophic lateral sclerosis? No, and for several reasons. To begin with, when I was examining his hands for the atrophy of muscles to which I have called your attention, I observed that there were some sores and scars on his fingers. Those scars and sores are of spontaneous origin, that is to say, they are not consequent upon any injury. We also find a scar on his right elbow, which, however, is the result of an injury. Let us next examine the man's eyes. Although his bulbar nerves are intact, when we come to examine the movements of the eyeballs we find he has marked nystagmus. You can see that he has definite nystagmus when he turns his eyes to either side. Moreover he has blunting of sensibility to pain and to thermal impressions on the whole of the right arm, and in certain other

parts. Now, it is not easy, owing to his mental condition, to show you this, but I will try to demonstrate it. I have succeeded in putting this pin under the skin without his being aware of it, which I think sufficiently shows that he is analgesic. I cannot test him in the ordinary way by asking him questions because of his mental state. There is also blunting of sensibility on the other side, but not to such a marked degree. We will next try him with regard to thermal impressions, by means of hot and cold test tubes. You see he cannot distinguish between the hot and cold tubes. He can nevertheless perceive tactile impressions, although he has this blunting to pain and to thermal impressions. So we have here this picture: that the man has spastic phenomena in the lower limbs, atrophy of muscles of the upper limbs, with nystagmus and analgesia and thermal anæsthesia. Another point we should like to know about would be as to the state of his back, that is to say, whether there is any curvature of his spinal column. As a matter of fact he shows very little defect in that direction. Commonly these cases show lateral curvature, some of them with kyphosis, some without, but he does not present anything noticeable in this respect. It is not a case of amyotrophic lateral sclerosis but one of syringomyelia. We have here a perfect picture of the affection. The history of the man's illness extends over ten years. The first manifestation he noticed was pain in his back and neck, radiating to the head and down the spine. He had this in paroxysms, and it continued for about six months. Then the pain ceased and he has had no return. At about the same time as the pain began he had some difficulty in walking. More recently he noticed some disability in connection with the left hand, but that condition apparently remained stationary. Soon afterwards, in the same year, he noticed some weakness of the right hand, but that defect remained comparatively stationary until two years ago, when the disability in his right hand became very much worse. Also, according to him, all the manifestations of weakness in connection with his legs and arms have been progressing up to the present time. Just before he came into the hospital he became depressed, and his mental condition was such that no history could be obtained from him on admission.

I now show you one other case to complete the

group of cases of paraplegia I selected for consideration to-day. This woman is twenty-two years of age. She presents spastic phenomena in connection with motor weakness in her lower limbs. The knee-jerks are exaggerated, and she has ankle-clonus on both sides. She has also the "extensor response" (Babinski's sign) on both sides. In short, she again presents the features of the case which we saw first this afternoon—spastic paraplegia with exaggeration of the tendon-jerks. She has no sensory defect in connection with her lower limbs. We examine her hands, but find no evidence of any muscular atrophy, or any other defect in them. We, however, ask her to touch the point of her nose to see whether there is any ataxy of her arms. At the end of the act you see a little intention tremor, which is more marked on the left side, although it is also present on the right. I will ask her to tell us a little of her history in order that you may hear her speak. All that is noticeable is a slight tendency to scanning speech. The defect seems very slight, but to the practised ear it is quite definite. With regard to the other points we wish to look for, as in the case of the man just now, we examine the eyes to see whether there is any nystagmus. There is nothing of a marked character in this respect, but she has nystagmus, though it is so slight that if you were to examine the case hurriedly you would not notice it. We have therefore found that she has slight, slow nystagmus associated with slight scanning utterance, intention tremor in the hands, and spastic phenomena in the legs. There are two other points in her case to which I must allude. When she phonates, that is to say, when she says "ah," her palate is drawn up more to the right than to the left side. In other words, she has weakness on the left side of the palate. Another point, which I cannot demonstrate to you, is that she has pallor of the optic discs, the left disc being paler than the right. These phenomena make up a picture of disseminate sclerosis. When we look back to the history of her illness we find some points which help us in arriving at this diagnosis. The patient had dragging of the left leg four years ago. During the first six months the leg continued to get rapidly worse, and then it remained stationary. Six months ago she began to have disability in connection with the right leg of a similar character to that in connection with the left, and during the same period she has had some shakiness of the hands. More recently she has had hesitancy with regard to micturition and precipitancy as regards defæcation.

THE CLINICAL DIAGNOSIS OF DIPHTHERIA IN RELATION TO TREATMENT.*

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IN recent years the methods of diagnosis of certain infectious diseases have undergone a remarkable development, so that whereas formerly we had to rely solely upon clinical facts for our diagnosis, we are now in a position to make a more exact diagnosis in these diseases by bacteriological methods. Among the most familiar instances of this stands the diagnosis of diphtheria. The bacteriological diagnosis of diphtheria, however, involves some delay, and it may be twenty-four hours, and sometimes more, before the practitioner receives a report of the bacteriological examination. In the meanwhile he has to rely on the clinical diagnosis in his conduct of the case. I propose to make a few remarks on the clinical diagnosis of pharyngeal diphtheria and on the indications we may draw therefrom, with regard to the immediate injection of antitoxic serum.

Bacteriological investigations have established beyond doubt certain facts in relation to diphtheria which have a very important bearing on the diagnosis of the disease. It has been shown that diphtheria may run its course without the formation of any true membrane in the throat, and on the other hand that membranous sore throats occur which are not dependent on the diphtheria bacillus. It has been further shown that while true diphtheria may run a most benign course, membranous sore throats caused by streptococcus infection may run a course not less malignant than true diphtheria. It is not wholly unnatural that, as a result of these investigations, an attitude of helplessness should have been assumed in some quarters with regard to the possibility of making a diagnosis of diphtheria from the clinical aspect of the case. Formerly a suspected throat was carefully scrutinised, every associated sign and symptom was carefully weighed, in order to arrive at least at a probable diagnosis. At present it is not unfrequently a case of a rapid glance at the throat,

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followed by the application of a sterilised swab, and the relegation of further consideration of the diagnosis until a bacteriological report has been obtained. This is for many reasons much to be regretted. I do not in the least undervalue the importance of the bacteriological examination, and I hold that in every suspected case it should be made as soon as possible, but while awaiting the result, I attach the utmost importance to the physician's taking every possible pains to make a diagnosis from the clinical signs and symptoms.

It is freely admitted that the clinical diagnosis of diphtheria has its limitations, but this is far from admitting that the clinical diagnosis has lost its importance, or that the bacteriological method should wholly supplant the clinical method. The clinical method enables us to affirm, in many cases with certainty, in others with very great probability, the nature of the disease. In some instances it supplements or confirms the bacteriological diagnosis, when the latter is not of a decisive character. It is upon the clinical aspect that we must rely in estimating the severity and probable course of the case, the degree of the diphtherial toxæmia, and the gravity of the septic complications. Above all, the clinical diagnosis is made on the spot, while the bacteriological diagnosis requires time; and, again, the facilities for obtaining a bacteriological report vary much in different localities and under different circumstances. The diagnosis of diphtheria at the earliest possible moment is of the utmost importance, and at the present day more so than formerly. Formerly, an early diagnosis was of importance, chiefly on account of the question of the isolation of the patient and the prognosis of the case. The early application of treatment was of less importance. It is true that different practitioners attached importance to this or that local remedy, while others set great store on the administration of this or that drug, but in truth there was no specific remedy known, and none the withholding of which for a day or two would have seriously impaired the chances of recovery. It is different now. If there is one thing more than another about the treatment of diphtheria with antitoxic serum on which there is a general agreement, it is the importance of employing the remedy at the earliest possible moment. As I have indicated, my chief object in making a few remarks on

the clinical diagnosis of diphtheria is to raise the question as to what rules we should lay down for ourselves, in the ordinary routine of private practice, with reference to the immediate injection of antitoxic serum in cases which we believe or suspect to be diphtheria, before a bacteriological examination has been made. It is an important question, and one in which, in my experience, the practice of medical men is far from uniform.

The considerations which must influence our practice in this matter will be, amongst others: (1) The importance we attach to the serum treatment, and especially to its employment at the earliest possible moment. (2) The reliance we place on the clinical signs and symptoms as elements in the diagnosis of diphtheria. (3) The probable course of the case, supposing it to be one of diphtheria. (4) The objections to the use of the serum, and the possible ill-effects that may arise from injecting it.

As to the first of these considerations I shall say nothing, because, I take it, we are all agreed on the value of the serum treatment, and on the great importance of its early employment.

As to the reliance to be placed on the clinical diagnosis, I have already expressed an opinion. It enables us to affirm with certainty in some cases the nature of the disease, while in others we are able to affirm it with a probability approaching to certainty. Such a diagnosis, however, can only be made upon careful examination of the case. Diphtheria presents itself under different types, and the diagnosis does not rest upon any one symptom, but upon a grouping of symptoms and upon a study of the case as a whole. No sign or symptom taken alone is reliable. We have to examine the throat in the best light obtainable, and the frontal mirror is often indispensable for this purpose. The condition of the lymphatic glands has to be noted. We have to consider the mode of onset, the character of the pulse and temperature, the general aspect and condition of the patient as well as other points in relation to the diagnosis. A history of exposure to infection will always carry weight. It is no part of my intention to enter into the various signs and symptoms which aid us in forming a diagnosis. These are to be found in books and are well known to most. Bacteriological discoveries have not materially altered their significance, nor have

they detracted from the value of the clinical pictures of diphtheria which are to be found in the older classical treatises on the disease.

One thing that bacteriology has done is to confirm the view which was already held by many, that diphtheria occasionally occurs in atypical forms, and to afford a certain means of making an exact diagnosis in such cases. This, of course, has been of enormous service. At the same time I think the frequency of cases of diphtheria which run their course under the guise of a simple non-membranous tonsillitis or pharyngitis has been somewhat exaggerated. One should always, of course, bear the possibility in mind when there has been of exposure to infection, and in the case of schools and other places where children are congregated it is wise to spare no pains, and to hold every sore throat suspect, until a bacteriological examination has been made, as a case of this kind may lead to a serious epidemic. Bacteriology has further taught us that certain membranous sore throats, with a more or less typical aspect of a diphtheria are not due to the diphtheria bacillus. These cases, however, are exceptional. It may safely be affirmed that in the vast majority of cases in which a competent observer makes the diagnosis of diphtheria the diphtheria bacillus will be found. I am aware that the experience of bacteriological institutes, where many specimens are examined, might, at first sight, seem to conflict with this view, as a very large proportion of the specimens sent fail to show the diphtheria bacillus. But it must be remembered that many of the specimens sent for examination are derived from cases which present little resemblance to diphtheria, but which, for various reasons, it was thought prudent to investigate.

Nothing that I have hitherto said will, I hope, be taken to mean that doubtful cases do not occur. Indeed, very many cases are met with in which the diagnosis, especially at the first visit, is extremely doubtful; cases in which not even a probable opinion can be expressed either way, and yet in these cases, also, we have to decide for or against the immediate use of antitoxic serum. It is in these doubtful cases that the third consideration, above mentioned, should, I believe, influence our course of action. Thus a case which we regard as suspicious may be very mild, and free from any disquieting symptom, a case which, even if diph-

therial, seems to be running a very benign course. Obviously in such a case we shall be disposed to wait for a bacteriological report. Another case which we consider doubtful may yet present symptoms which make us feel that if it is diphtherial it will prove more or less grave. Waiting for a bacteriological report in such a case may mean waiting until the patient is *in extremis*, and it will seem more prudent to inject the antitoxic serum at once.

The fourth consideration, namely, the objections to the use of antitoxic serum cannot be lightly passed over. There is the sentimental objection which parents have to the little operation which administration of the serum involves, and certainly any doctor who promiscuously injected his patients in cases which ultimately proved to be non-diphtherial would acquire the reputation of an alarmist. Antitoxic serum, moreover, does produce, in certain cases, symptoms which, though they weigh very little as against the benefits derived from the treatment, cannot be entirely ignored. Thus an urticarial or erythematous eruption may appear within a few days of the injection, which may be accompanied by a slight elevation of temperature. Such an eruption is generally very transitory, lasting from a few hours to a day or two, and is accompanied with little or no general disturbance of the system. At a somewhat later period, about the twelfth or fourteenth day, symptoms of a more severe character are occasionally observed. They may be ushered in by a sharp rise of temperature, and sometimes by vomiting. Articular and muscular pains, not unfrequently of a severe character, may follow, as well as a more or less generalised eruption of a scarlatiniform, measly, or erythematous type, which may be accompanied by intense pruritus. Although these phenomena subside in three or four days without leaving any ill-effects, they may, while they last, cause a good deal of discomfort to the patient, and a certain amount of anxiety to those around. Then again, one or two sudden deaths have been recorded after the injection of the serum, and however they may be interpreted, the publicity which has been given to these accidents has not failed to awaken a repugnance to the promiscuous use of the serum.

I shall now endeavour to formulate certain principles which, in my opinion, ought to guide us in the

use of antitoxic serum whenever we find ourselves in the presence of a case which, from its clinical characters, we regard as an undoubted or suspected case of diphtheria.

1. In every instance in which the clinical examination assures us that the case is one of diphtheria the antitoxic serum should be injected as soon as possible. No case which, from its clinical aspect, we regard as undoubted diphtheria, however benign it may seem to be, should be excepted, as we are never sure that the case may not take on a more severe course, or that the disease may not propagate itself to the larynx. Even if a case is already well advanced, although we may not expect the good results which follow the early employment of the serum, it should be given a trial. A beneficial result is always possible.

2. In a case in which the diagnosis is doubtful, if the symptoms are of such a nature that, supposing it to be a case of diphtheria, we should anticipate a severe course, an injection should be made at once, without waiting for the bacteriological report. Thus we may have a case in which the throat is by no means characteristic, and yet we may discern symptoms which we are accustomed to associate with diphtherial toxæmia (pallor, frequency and feebleness of pulse, apathy, prostration); or the case may be obviously of a severe septic character, and although we do not anticipate any influence on the septic organisms from the antitoxic serum, yet in a disease in which two infections are combined it is something to control the effects of one of them, and as such cases are likely to run a severe course, we should give the patient every chance by injecting the serum at once. In the same way, if a croupy cough, or husky voice, or any symptom of laryngeal trouble is present in a case in which the diagnosis of pharyngeal diphtheria is doubtful, we should inject antitoxic serum without waiting for a bacteriological examination.

3. In a doubtful case in which the symptoms are obviously mild, we may wait until we receive a bacteriological report. In the interval, however, the patient should be strictly watched, and if signs occur which render the diagnosis of diphtheria no longer doubtful, or which indicate a severe course in the event of its being diphtherial, especially any laryngeal symptoms, the serum should be injected without further delay.

4. In all cases of croup in children in which the pharyngeal signs of diphtheria are wholly absent, unless we can exclude the possibility of primary laryngeal diphtheria, either by laryngoscopic examination or otherwise, it is best to inject antitoxic serum at once. I know of more than one case in which neglect of this rule has given cause for regret.

It is impossible to frame rules which will meet every case, but I think the foregoing will meet the majority of cases which occur in practice, and may be safely followed. The early injection of the antitoxic serum is, I believe, of immense importance, and a delay of even twenty-four hours may make a serious difference in some cases. Hence the importance of the clinical diagnosis. The clinical signs afford valuable diagnostic indications in diphtheria apart from bacteriological examination, and it is only by a proper appreciation of the clinical signs that we can arrive at a right decision as to the immediate injection of the antitoxic serum.

Simultaneous Administration of Calomel and Iodides.—Calomel applied locally or ingested while the subject is under the influence of potassium iodide previously or simultaneously administered, forms with the iodine a caustic combination—the iodide of mercury—leading to abscess formation, ulceration, etc. The iodine ingested in the form of potassium iodide is eliminated in the secretion on every mucous membrane, and when it comes in contact with calomel, the caustic compound that results is liable to form an ulceration, in the depths of which the yellow crystals of the mercury iodide are plainly visible. Rabbits treated with potassium iodide, and then receiving a stomach injection of calomel, exhibited numerous ulcerations in the gastric mucosa, surrounded by a hyperæmic zone showing the irritating action of the salt. These caustic ulcers are liable to occur in any stomach after ingestion of calomel when the system is under the influence of potassium iodide. The physician should ascertain whether the subject has been taking potassium iodide before prescribing calomel, either internally or for local application on any mucous membrane. Calomel is the best test for iodine in the organism. The simplest application of the test is to add a little calomel to the saliva. If it contains traces of iodine the calomel turns yellow.—*Journ. A.M.A.*, Jan. 11th, 1902.

A CLINICAL LECTURE ON MITRAL REGURGITATION.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, November 20th, 1901.

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(Concluded from p. 240.)

Physical signs.—These we may perhaps best consider in the following order :

1. *Inspection.*—In a case of mitral regurgitation, if the heart is but little hypertrophied, inspection will not help us much. If, however, the disease be more serious, and the heart greatly enlarged, then, on examining the chest, we may be at once struck by something abnormal ; we may see *obvious systolic retraction of the intercostal spaces over the cardiac area*. This occurs most frequently in children, and is most marked in the fourth and fifth spaces in the region near apex. It is not uncommonly assumed that such retraction indicates pericardial adhesions. This sometimes may be the case, but in general, as Sir William Broadbent has pointed out, it is the result merely of atmospheric pressure, the hypertrophied heart diminishing rapidly in bulk at each systole, and thus allowing the walls of the chest to yield to the pressure of the atmosphere.

Another point, also, which should always be noted on inspection is the *condition of the veins in the neck*. In certain cases these will be found dilated, and sometimes also they will be found to pulsate. It should be noticed, however, that this dilation generally disappears either entirely, or at least to a great extent, when the patient takes a deep inspiration. This is a point of much importance, since in cases of aneurysm or intra-thoracic growth when the superior vena cava is compressed, although the veins may be greatly dilated, yet their calibre does not diminish as a result of inspiration. The effect of a deep inspiration upon the dilated veins in the neck may thus become a point of considerable importance in diagnosis.

2. *Palpation.*—Let us now pass to palpation. By palpation we are able to localise with exactness the *position of the apex-beat*. Where is this in mitral regurgitation ? Is it displaced merely outwards, or is it displaced downwards as well ? Upon this point authorities are not always agreed, and I

have, therefore, carefully analysed the position of the apex-beat in thirty-three of my cases. The following is the result :

TABLE V.—*Showing the interspace in which the apex-beat was felt in thirty-three cases of mitral regurgitation.*

	No. of Cases.
Fourth space	1
Fifth space	22
Sixth space	9
Seventh space	1

Given in more detail, it may be stated that the apex-beat was in 1 case in the fourth space, in 22 in the fifth space, in 9 in the sixth space, and in 1 in the seventh space. Further, of the 22 in which it was in the fifth space, it was displaced outwards in all but 3 (in 11 being in the nipple line and in 8 outside it), while of the 9 cases in which it was in the sixth space it was displaced outwards in all. We may say, then, that *in about two thirds of the cases the apex-beat was displaced outwards only (being in the fifth space), and in one third it was displaced downwards as well as outwards. (In one eleventh of the cases the apex-beat was in the natural situation.)* It may be added that simple displacement outwards is commonly the result of dilation and hypertrophy of the right ventricle ; while when the apex-beat is displaced downwards as well, a similar affection of the left ventricle is also indicated.

By palpation, however, we may occasionally demonstrate something more than the mere position of the apex-beat ; we may recognise in addition the presence of a *thrill* in this region. This, as you are well aware, is systolic in time, and synchronous with the cardiac shock, not occurring immediately before the latter, as is the case in mitral stenosis. In my experience such a systolic thrill is distinctly uncommon in mitral regurgitation. In two only of my thirty-three cases did I observe it.

3. *Percussion.*—Percussion shows us that in all but the mildest cases of mitral regurgitation the cardiac dulness is augmented. As a rule, the dulness first increases upwards, reaching, perhaps, the third space or third rib. Soon, however, it increases to the right, and later this extension becomes very marked, the dulness reaching perhaps an inch or an inch and a half beyond the right hand border

of the sternum. As a result the cardiac dulness assumes the quadrilateral shape with which we are all familiar. Later still, when all the cavities of the heart have become dilated, the area of dulness enlarges in all directions, upwards, to the right, and also to the left. The shape of the dull area then becomes roughly globular, resembling very closely that of a large pericardial effusion, and in some cases the difficulty in diagnosing between the two conditions may become extreme.

4. *Auscultation*.—This method of examination reveals to us the cardinal sign of mitral regurgitation, namely, the systolic murmur, heard best at the apex, conducted into the axilla and heard also posteriorly between the spine and the angle of the left scapula. Concerning the character of this murmur I need not say much. You are all familiar with it. It generally possesses a blowing quality, and is sometimes soft and sometimes loud. Occasionally, however, it presents a characteristic of more importance, in that it may be musical in quality. This I noticed in two out of my thirty-three cases. The importance of this quality we shall consider later.

Another physical sign of some little importance often heard on auscultating a case of mitral regurgitation is *accentuation of the pulmonary second sound*. To be of any diagnostic importance, however, this accentuation must be marked and persistent, since there seems to be little doubt that normally the pulmonary second sound is in fact louder than the aortic (Vierordt). But if the accentuation really is marked the sign is of importance, for it proves that the tension of the blood in the pulmonary system is greater than usual, thus indicating some obstruction to the flow of blood at the mitral orifice.

The pulse.—Let us now pass to the pulse, and in considering this subject the great question that presents itself is this: Does the "typical mitral pulse," in which the pulse is irregular both in force and in rhythm, occur in every case of mitral regurgitation? To this question a decided negative may be given. It is indeed true that if the lesion is serious in the vast majority of cases this alteration in the pulse will be found, but in mild cases, in which the reflex is but slight, the pulse may remain perfectly regular. In ten recent slight cases, in which I have specially noted this point, I found that the pulse was regular in six and irregular in four. The figures are too small to be of

value in themselves, but they emphasise the point that in mild cases the pulse is not uncommonly quite regular, and, I may add, not more than slightly quickened.

Diagnosis.—As regards the diagnosis of mitral regurgitation, it may be at once conceded that if the lesion is a serious one, if the apex-beat is displaced, if the pulmonary second sound is accentuated, the heart hypertrophied and dilated, and a marked murmur present, there can be little or no question as to the diagnosis of the disease. The difficulty arises rather in the slighter forms, when a systolic murmur may be heard in the apical region, without there being any other evidence of valvular disease. In such cases we have to consider whether the murmur is really produced within the heart, or whether it may not be, in fact, a *cardio-pulmonary murmur*, a class of murmur especially studied by Potain. As you are aware, a cardio-pulmonary murmur indicates a murmur produced not in the heart, but in the lung overlying the heart. When the latter organ contracts the thin edges of lung overlying it become compressed, and under certain circumstances the air contained therein is quickly driven out; in this way, at each beat of the heart, a systolic murmur is produced, which to the ear closely resembles the endocardial murmur of mitral regurgitation, and from which it is often extremely difficult to diagnose it. How, then, are we to distinguish the two? In the first place we should notice carefully the situation of the murmur. If the murmur is endocardial it will be heard loudest exactly over the situation of the apex-beat. If, on the contrary, it is a cardio-pulmonary murmur, it will often be best heard not exactly at the apex, but slightly to the right or slightly to the left of this position. Secondly we should notice the effect of posture upon the murmur, cardio-pulmonary murmurs being more influenced by change of position than those due to organic disease. Thus exocardial murmurs are often more evident in the recumbent position than in the erect, while on one occasion at least I have known the reverse to be the case. The chief point, however, upon which we must rely in making a diagnosis between the organic and the cardio-pulmonary murmurs is the fact that the latter are influenced to a remarkable extent by the respiratory movements, though the exact alteration produced is not always the same in every instance. In the majority of cases the

murmur is increased in loudness when the patient takes a deep inspiration, and not uncommonly is diminished, or even becomes inaudible, at the end of a deep expiration. If, then, in a given case a murmur is found to possess such characteristics, this must be regarded as strong evidence in favour of the view that we are really dealing with a murmur produced in the lung, and not with one originating in the heart.

Prognosis.—In heart disease the prognosis depends to a great extent upon the nature of the lesion. As you will no doubt remember, Peacock was of opinion that aortic regurgitation was the most serious lesion, then came mitral regurgitation, then mitral stenosis, while least serious of all, in his opinion, was aortic stenosis. Lately, however, exception has been taken to these views, Sir William Broadbent, for example, believing that mitral regurgitation is the least serious of all the valvular lesions. On this point it is extremely difficult to speak with certainty, the prognosis depending to so great an extent upon the severity of the lesion. Nevertheless it is true to say, as Broadbent points out, that if the lesion is slight the patient may live his whole life and do his ordinary work but little inconvenienced by his valvular disease. One such case illustrating this fact I bring before you this afternoon. The patient is a female, æt. 52. She had rheumatic fever when 15 years of age, a second time when 16, and again for the third time when 17, but has never had it since. In 1869, when 18 years old, she was seen at St. Bartholomew's Hospital by Sir William Church, who, so she tells me, stated that her heart was then affected. In spite of this, however, she is now alive and well, though at the present time her lesion is a somewhat serious one. The apex-beat is in the sixth space in the anterior axillary line, and the pulmonary second sound is accentuated, but the heart and pulse are not very rapid, beating only ninety-six to the minute. The pulse is a little irregular. I should add that there is at the present time a faint pre-systolic in addition to the systolic murmur. This patient, then, has performed her ordinary duties and gone about her ordinary work during a period of thirty-two years, in spite of her valvular disease, and even at the present time, with the exception of a certain amount of shortness of breath, she does not complain of anything very serious.

The prognosis, however, in a case of mitral re-

gurgitation depends for the most part upon the extent of the lesion. How may we estimate this? Can we effect it by noticing the character of the murmur? The loudness or softness of this sound gives us, of course, no indication of the severity of the disease, but there is one characteristic sometimes noticed about the murmur which is of some assistance; I mean the possession by it of a *musical character*. When this is heard it often indicates slight disease, the sound being produced by the strongly-acting heart propelling a fine but forcible jet of blood into the auricle through a mere chink in the centre of the mitral orifice. Unfortunately, however, this rule is not an absolute one, for though a musical mitral murmur most often indicates a low degree of regurgitation, yet the same sound may be also sometimes heard in cases of much greater severity. In deciding, then, upon the extent of the lesion, it is evident that we must not rely to any great extent upon the character of the sounds of the heart. Our opinion, rather, must be based upon the evidence which exists of hypertrophy and dilation of the organ, the increase in size of the heart being a measure of the extra strain thrown upon the circulation by the valvular disease. Thus if in a given case the apex-beat be displaced downwards and outwards, if the pulmonary second sound be accentuated, and the heart greatly dilated and hypertrophied, then we know at once, whatever the character of the murmur, that the lesion is serious, even although compensation may for the time have been perfectly effected. In such a case, also, our prognosis would be serious, since we know by experience that a compensated heart is prone to give way whenever some extra strain is thrown upon the organism.

There is, lastly, one other point which we may consider in connection with the question of prognosis, and that is the *danger of sudden death* in a person suffering from mitral regurgitation. As a matter of fact, in this form of valvular disease such a termination is excessively rare. This is well shown by some statistics collected by Dr. Horder* from the patients who were brought in dead to St. Bartholomew's Hospital during the five years 1894—1898. These amounted in all to 126 cases. Among them the fatal event was in 22 the result of heart disease, aortic disease being the

* 'St. Bartholomew's Hospital Reports,' 1899.

cause of death in 10, while mitral regurgitation was responsible for 1 case only. It is therefore only most rarely that sudden unexpected death occurs in this variety of heart disease.

I have so far spoken of the prognosis of mitral regurgitation when the latter is due to organic disease of the valves. In those cases where the reflux is due to failure of the muscular tissue of the heart, the prognosis must vary with the cause. In a case of anæmia the prognosis will be good, and the heart will probably quite recover. If, however, the regurgitation be due to fibroid, syphilitic, or fatty disease of the heart, then the prognosis is far more serious.

Relation of mitral regurgitation to phthisis.—Rokitansky, as you will remember, held that all forms of disease of the heart or lungs which induced cyanosis were incompatible with tuberculosis. He believed, therefore, that mitral regurgitation and stenosis were practically antagonistic to pulmonary phthisis. It is by no means uncommon, however, at this hospital to see patients which demonstrate both lesions,* and during the last two years I have had here under my care four such cases. Three of them were examples of mitral regurgitation, combined with pulmonary tuberculosis, while the fourth was a case of mitral stenosis with a similar lesion in the lung. In all four tubercle bacilli were present in the sputum. In two of them there was a definite history of rheumatic fever, and in a third "joint-pains" had been complained of. The fourth case showed no evidence of previous rheumatism. Although, therefore, Rokitansky's view cannot be accepted in its entirety, it would be a mistake, nevertheless, to regard it as wholly untrue, for this much we may affirm: that in cases in which mitral disease is present the tuberculosis, as a rule, runs a somewhat more chronic course than usual, and therefore a fairly good prognosis as to duration may be given. In the four cases to which I have referred, and of whom two, I hope, will be present to-day, the pulmonary disease has undoubtedly been of a comparatively quiescent type. Three of the patients are still alive and doing fairly well, while the fourth, although the disease was itself of the chronic variety, died unexpectedly of hæmoptysis.

Treatment.—In considering this all-important

question, the first point to be remembered is that the mere diagnosis of mitral regurgitation does not necessarily indicate the administration of digitalis. To give the drug in all cases, as is sometimes done, is not only useless but also harmful, for, unless the heart requires it, no benefit is derived at the time, while, on the other hand, the patient is accustomed to a drug which later on might have been of inestimable value to him. The indications for the use of digitalis, then, are irregularity of the pulse and heart, the onset of œdema, and the diminution in the amount of urine, in short, evidences of a failing heart. Given under these circumstances digitalis is often invaluable. The heart (both right and left sides) is stimulated, the rate of beat is diminished, while the force of contraction is increased. The amount of urine also is augmented. As a result of these beneficial changes the œdema often diminishes and disappears, and compensation is re-established. If there be much dropsy, however, it may be well to prescribe citrate of caffeine in addition to the digitalis. Citrate of caffeine (gr. iv) with tincture of digitalis (℥xij), given every four hours, sometimes produce marvellous results in cases of this kind. Thus I have known the amount of urine increased from 26 ounces to 86 ounces on the following day, then to 72, and finally on the fourth day to 100 ounces, the œdema and hydro-thorax at the same time disappearing, and compensation being gradually re-established. Sometimes, however, digitalis fails, and this is especially the case when the patient has been treated with it before for similar attacks of heart failure. Other drugs, such as strophanthus and convallaria, must then be tried, but only rarely do they have an effect approximating to that of digitalis. In all cases, also, whatever the cardiac tonic prescribed, we must always remember to see that the bowels are kept well open.

The administration of cardiac stimulants is, however, not the one and only method of treatment at our disposal. In certain cases *venesection* may be of the utmost value. This is especially indicated if the right side of the heart is over-distended, if the patient is cyanosed, and if the pulse is highly irregular and beginning to fail. Ten ounces of blood then removed will sometimes, as it were, give the patient a new lease of life, the previously over-distended right ventricle being enabled once more to contract efficiently, and duly perform its

* See Dr. Percy Kidd's important paper on "The Association of Pulmonary Tuberculosis with Diseases of the Heart," 'St. Bartholomew's Hospital Reports,' 1887.

functions. In some cases it may be difficult to bleed; wet-cupping or leeches may then be tried, and may prove to have a similar beneficial result. Venesection, then, in the later stages of mitral regurgitation should always be borne in mind as a very valuable adjunct in certain cases to drug treatment.

It may be asked, however, if our drugs fail us, whether there is no other method of treatment which we can try? Fortunately there is. In such a case the *Nauheim treatment* may be recommended; the patient, that is to say, may be ordered a course of baths (containing sodium chloride and calcium chloride in due proportions,* and also a certain amount of carbonic acid), while, if desired, these may be combined with a course of exercises, performed slowly and against resistance. As a result of this treatment it sometimes happens that compensation which has failed, and which it has been impossible to restore by the action of tonics such as digitalis or strophanthus, becomes again re-established. When at Nauheim, six years ago, I was much impressed by a case of this kind. The patient was a man of about forty-five years of age, who was suffering from aortic and mitral regurgitation with marked dropsy and hydro-thorax. He had been treated at home with digitalis for some months, but with no benefit. He was then sent to Nauheim (two and a half litres of fluid being withdrawn from his pleural cavity in order to permit of his travelling). At Nauheim he took the baths and almost at once began to improve. So marked, indeed, was the improvement, that at the end of a four weeks' course, when I examined him in Dr. Schott's consulting room, the hydro-thorax had disappeared, the dropsy had gone, and the heart was now beating fairly regularly. Compensation, in fact, for all practical purposes, had been re-established. The patient himself, I should add, volunteered that he felt "younger now than he had done for twenty years." This case is not an isolated one, similar instances of recovery being well known. If therefore, in a given case, drugs fail in their effect, the "Nauheim treatment" (either baths alone or combined with exercises) should certainly be tried, though improvement so marked as in the case recorded above must not be expected in every instance. Should it be possible, the patient should undoubtedly be sent to Nauheim, there to undergo the treatment, for although the baths may be

artificially prepared in England,* yet the patient loses the psychical effect of the change of climate and of scene which undoubtedly exercise a beneficial action in such cases.

There is, lastly, one further point which I desire to consider with you, and that is the question of *residence* and of winter climate. Where then should a patient with mitral regurgitation live? This is a question which is not uncommonly asked. No special place need be laid down. But we may say that any locality which is dry, which receives a good deal of sunshine, and which is not exposed to violent winds, is suitable for such a case, for here the liability to further attacks of rheumatic fever and to secondary bronchitis would be greatly diminished. Many districts answer to this description, and will suggest themselves to you. From personal knowledge, however, I might suggest two: Woburn Sands (Apsley Guise), on the green sand area of Bedfordshire, and Malvern, the latter combining to some extent the attractions of a town with the advantages of the finest country air.

In conclusion I propose to say a few words as regards *winter climate*. The majority of our patients—those, at all events, whom we see in hospitals—will probably be obliged to stay during the winter months where they have lived throughout the year, but to a certain class it is possible to winter elsewhere. Where, then, should such patients be advised to go? One climate offers itself which is exceptionally suited to their requirements, that, namely, of Egypt. In this country, Cairo, in no wise suited to the invalid, should be avoided. Patients should be sent to one of the four health resorts, where the pure air of the desert can be experienced. These resorts are Mena House and Helouan, not far from Cairo, and Luxor and Assouan, on the banks of the Nile, much further south. The great characteristics of the desert climate which may be experienced in varying degrees at these health resorts, and which are so beneficial in cases of the kind which we are now discussing, are well shown forth in the following table, to which the similar figures for

* For the method of artificially preparing the baths, and for a description of the exercises used, see the appendix to the third edition of Sir William and Dr. J. Broadbent's 'Heart Disease,' 1900.

TABLE VI.—*Meteorological Observations for the four months December to March at the following Egyptian and English Stations.*

	Years to which the data refer.	Temperature.				Relative humidity 9 a.m. Per cent.	Rainfall.	
		Av. max.	Av. min.	Av. mean.	Av. daily range.		Days.	Inches.
Mena House	1895-96	70.4°	48.7°	59.5°	21.7°	77.4	15*	1*
Helouan	1894-96	71.7°	49.4°	60.5°	22.3°	59.4	15*	1*
Luxor	1893-96	78.1°	49.6°	63.8°	28.4°	56.8	1	Trace.
Assuan	1893-96	82.1°	54.5°	68.3°	27.6°	50.5	0 or 1	Trace.
Malvern	1893-96	44.5°	34.4°	39.4°	10.1°	85.1	61.4	7.9
Regent's Park	1881-89	44.7°	34.2°	39.5°	10.5°	86.7	54	7.3

* Approximate.

Malvern and Regent's Park have been added for comparison.*

From these figures it will be seen that in the first place the temperature in the winter at the Egyptian health resorts is a good deal higher than is the case in England, ranging from 59.5° to 68.3° as compared with 39.5° and 39.4° in London and Malvern. In the second place, the rainfall is very different. In England this varies from 7.3 to 7.9 inches during these four months and falls on between fifty and sixty days. At the Egyptian health resorts it measures at most one inch and falls on never more than fifteen days. As a result of this diminished rainfall and the exceptional dryness of the soil the relative humidity (or percentage of aqueous vapours in the air) becomes extremely low. In England at 9 a.m. this is, at Malvern, 85.1° and at Regent's Park 86.7°, while at the Egyptian health resorts it varies between 77.4° and 50.5°. As regards their temperature, rainfall and humidity, the Egyptian health resorts thus enjoy great advantages, but further they stand pre-eminent in the amount of sunshine which they enjoy. In Egypt, during the winter, sunshine is almost constant. In England, on the contrary, this is unhappily not the case. Thus at Apsley Guise (Woburn Sands) during the four winter months, it only amounted to 21.5 per cent. of the possible

total, while at Kew 226 hours were registered, or 19.1 per cent. of the possible amount. As a result of these characteristics of its climate Egypt may be warmly recommended to those sufferers from mitral disease who desire to winter abroad, for here their frames will be invigorated by the brilliant sunshine and the stimulating air, while the dryness of the atmosphere will diminish their liability to bronchitis or to secondary attacks of rheumatic fever.

Pregnancy after Nephrectomy (STEINHEIL).

—Five years after one kidney had been extirpated on account of a tubercular affection, the patient, a woman of 25, apparently in good health, but rather pale, consulted Steinheil in regard to a commencing pregnancy. She had been warned of the danger of conception in her case, and he hesitated whether to induce an abortion or to let the pregnancy take its course, deciding finally on the latter. The urine contained pus and was slightly blood-stained. The pregnancy terminated normally and the patient recovered, with only the drawback that permanent catheterisation was necessary for a short time. The child is healthy, and the mother remained in good health for twenty-one months, when she succumbed to the extension of the tuberculous process. Whether she would have survived longer if the pregnancy had been interrupted is an open question.—*Journ. A.M.A.*, Jan. 11th, 1902.

* The data for the Egyptian resorts have been taken from Dr. Leigh Canney's admirable work 'The Meteorology of Egypt and its Influence on Disease,' 1897; the humidity figures for 9 a.m. representing the mean of those given by him for 8 a.m. and 10 a.m. respectively.

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OPERATIVE TREATMENT OF FRACTURES.

Inaugural Address delivered at the Opening Meeting of the Surgical Section, Royal Academy of Medicine, Ireland.

By THOMAS MYLES, F.R.C.S.I.,
President of the Royal College of Surgeons in Ireland.

GENTLEMEN,—To-night I purpose bringing under your consideration a few points in connection with such a comparatively commonplace subject as injury to the long bones.

In recent years visceral surgery has perhaps too exclusively occupied the attention of operators, and with the brilliant result of such concentration of effort you are all familiar, but I think that in the interval perhaps an insufficient amount of attention has been devoted to the consideration of the subject that I now intend to place before you. While in the surgery of the thorax, abdomen, and head the enormous advantages of an aseptic technique have been fully availed of, we have, I feel, been rather remiss in applying the same principles to the treatment of fractured bones. It seems almost as if the boldness and enterprise which have achieved such remarkable results in abdominal surgery are considered entirely out of place when applied to the treatment of fractures. It still is considered compatible with good surgery that a fracture of the shaft of the femur should be followed by a shortening of from one to two inches, and the man who, without a moment's hesitation, would open the abdomen to seek for a possible perforation of the stomach or intestine, shrinks from the much smaller operation of exposing the broken ends of the shattered femur and securing perfect coaptation of the broken surfaces by the application of simple mechanical appliances. This, I think, is hardly creditable to us, and shows that notwithstanding all our talk about the enterprise and boldness of modern surgery, our minds are very apt to run in a groove, and with difficulty do

we extricate ourselves from the fetters of tradition. On no other hypothesis can our adherence to present methods be explained. At a time when aseptic surgery was in its infancy there may have been some justification for the unwillingness of surgeons to undertake what seemed great risks, but now, when the principles of aseptic technique are thoroughly understood, and every modern hospital is equipped with the means of carrying out this technique thoroughly, we are no longer justified in trusting to what may without disparagement be spoken of as methods of haphazard, instead of boldly using those of scientific accuracy. I am not advocating that every case of simple fracture should be subjected to operation. Ordinary fractures, for instance of the tibia or fibula, unaccompanied by displacement, usually give excellent results when treated intelligently by the methods hitherto in vogue, but it does seem rather a reflection upon us that we should still regard with equanimity a shortening of the limb amounting to one and a half inches as the result of a fracture of the shaft of the femur, or be content with the permanent lameness and loss of function which so often accompany a Pott's fracture. Every surgeon is aware of the difficulties of treating a fracture involving both bones of the forearm or separation of the lower epiphysis of the humerus. In both these instances deformity and impairment of function are the too frequent results of our present methods, and I would plead, therefore, for a careful consideration of these methods and an inquiry as to the possibility of improving them.

The surgical world owes a great debt of gratitude in this connection to the labours of Mr. Arbuthnot Lane, who has for many years past laboured with but indifferent success to impress his own bold and sagacious views upon the general body of the profession. In America, where traditions are of less influence than they are with us, his suggestions have met with a ready acceptance, but it must be confessed that in Ireland, as yet, little effort has been made to tread the path he has pointed out to us. It is true that in occasional cases, such as ununited fractures, or those resulting in angular union, we adopt modern methods, but I think these isolated instances do not relieve us of the reproach that in this particular branch of surgery Dublin lags behind, instead of being in the van, as it ought to be.

My purpose to-night is not to dilate upon this subject at large, but rather to confine my attention to one or two cases illustrative of the subject under review, and from them to make any larger generalisations that may seem indicated.

Quite recently two cases of rather rare injury were under my care at the Richmond Hospital, and were both submitted first to examination by the X rays, and subsequently to operation. They were both cases of fracture of the neck of the humerus with dislocation of the head towards the axilla. As this particular injury is not discussed at any length in the ordinary text-books, and as both the skiagraph and operation taught me a good deal I did not know before, I feel that no apology on my part is necessary for bringing the subject under your notice to-night. As you will see later on, the fundamental question that I have raised in the first part of this paper was forced upon my attention in these two cases.

My first case was that of a woman *æt.* 40 years, a patient in the North Dublin Union Infirmary, sent to me by Dr. Caleb Powell, a Fellow of this College. The accident had occurred some months before, and a diagnosis of dislocation, with some other indefinite injury to the upper end of the shaft, was made. The shoulder-joint was ankylosed and the arm practically useless. An X-ray examination by Dr. R. Lane Joynt showed that, in addition to the dislocation, there was a fracture of the anatomical neck without impaction. As there was both pain along the nerve-trunks and cedema of the hand and arm, and as the limb was practically useless, I determined to operate. This I did by an anterior incision, such as is usual in excision of the head of the humerus. The operation in this case, as in all other cases of old-standing uncomplicated dislocations, was very difficult, much more so than is usually found to be the case in resections for disease of the bone. This difficulty was due to the altered relations of the parts, to the thickening of the capsule and ligamentous structures around the joint, and to the general shrinkage which takes place when a dislocation has existed for any length of time. To permit sufficient freedom of movement the long head of the biceps had to be divided and the capsule split very freely. When at length a good view of the parts was obtained, I found that the head of the humerus along its posterior narrow edge was

attached by osseous union to the anterior edge of the glenoid fossa and a narrow area of the venter of the scapula adjacent thereto. On the anterior edge of the glenoid fossa, encroaching on the cartilage, was a distinct crescentic depression, into which the head was imbedded—apparently the pressure of the latter had first set up a rarifying osteitis, followed later on by osseous union between the apposed parts. The bond of union had to be divided with a bone forceps before the head could be removed. Portion of the great tuberosity was removed also, and the case became one simply of resection.

My second case was that of a man æt. 42 years, who fell about fourteen feet on to his head and shoulder. He was at once brought to the neighbouring hospital, where a diagnosis of dislocation was made and vigorous repeated efforts were made under chloroform to reduce it. It is impossible for me to say if the fracture and dislocation co-existed from the outset, or if, as he alleges, the fracture was produced by the vigorous efforts at reduction. I am inclined to think, from the nature of the accident, a fall from such a height, that the two injuries were produced at the same moment, and that the repeated efforts made to effect reduction under anæsthesia were made after due recognition of the nature of the lesion. This is rendered more probable by the fact that when he came under my care some months later even then crepitus was easily obtained, and must have been still more easily obtained a few hours after the accident.

On examination of the injured area a large bony mass could be felt in the shoulder extending inwards beneath the coracoid process; this was smooth, and felt like the head of the humerus. There was but little flattening of the deltoid or prominence of the acromion. The circumference of the shoulder was nearly one inch greater than on the opposite side. He had practically no power whatever in the arm, and any movement caused pain. An X-ray photo taken by Dr. R. Lane Joynt showed that the head of the humerus was dislocated inwards, and that the main line of fracture was through the great tuberosity. In addition, a long splinter was detached from the outside of the shaft, extending for some three inches downwards.

This case I also submitted to operation, but I found it necessary, in addition to the long anterior incision, to divide part of the deltoid close to its

insertion, so as to obtain sufficient room for the subsequent manipulations. I confess I was somewhat astonished on exposing the parts to find the head firmly ankylosed to the scapula, as in the former case. The seat of attachment, however, was lower down on the scapula, and much further inwards. It was not a mere line of bony union, as in the first case, but the posterior edge, and nearly half of the articular surface, was, as it were, imbedded in the scapula and incorporated with it. All attempts to displace it were futile, and I had to remove it piecemeal with cutting forceps. On the other hand, the long splinter of bone was quite ununited, and showed no attempt at bony union. This is certainly not what one would have expected from *a priori* reasoning.

One of the dangers of this injury is said to be necrosis of the head, but in my two cases at least experience does not support the theories based on *a priori* reasoning. It is undoubtedly a strange fact that the detached head, instead of necrosing, should have become firmly united to the scapula, while the long splinter of the shaft was still unattached. In this case, also, I had to resect a part of the great tuberosity so as to obtain a smooth end to the humerus.

These two cases are, to my mind, typical illustrations of my thesis, that the principles of modern surgery are not applied to the treatment of fractures with anything like the same boldness that they are applied to other surgical ailments.

First comes the ever-present difficulty of diagnosis. I think I am justified in making the assertion that ordinary methods of examination are not sufficient to enable even the most experienced surgeon to make an accurate diagnosis of these complicated injuries about the shoulder. Every Irish surgeon is bound to speak respectfully of the work done by the late Professor Robert Smith, but no one, I think, now-a-days will be found to assert that the admirable rules he has laid down for the differential diagnosis of injuries about the shoulder cover the entire ground, or are in themselves sufficient to enable an ordinary surgeon to make an absolutely certain diagnosis.

To meet the difficulty of teaching students, the teacher is compelled to adopt a system of classification which divides these injuries into separate groups, and to endeavour to place any one injury in some one of these groups. Hence, as a logical

consequence, has arisen the unnatural divisions of these fractures into intra- and extra-capsular, etc., with or without impaction. The learner, with his much smaller experience, is still more urgently impelled in the same direction, and when called to examine one of these cases his first mental effort is to place it in group A or B. Now this dogmatic teaching is, I believe, responsible for very many errors in diagnosis, and I am convinced that if men could approach the questions before them with a more open mind, unhampered by the venerable dogmas of the past, such errors would be less frequent. Nature does not care much for our classifications, and is not withheld by respect for surgical traditions from superimposing a dislocation upon any or every fracture.

The first requirement in surgery is accurate diagnosis, and I do not believe that the precise rules laid down by the great teachers in the past are sufficient in themselves to supply what we require. Every one of us has had experience of the great difficulties that attend the diagnosis of such injuries, and I hope I will not be thought to labour the point when I say that such injuries should not be treated at all until either an X-ray photograph has been made by a competent person, or a thorough examination has been made under an anæsthetic. The former course is far the more reliable and informing, but it is not always available. Even when an X-ray photograph has been taken it requires skill and experience in such work to read aright the lesson it conveys. We must not fail to remember that such injuries may be very complicated, several fractures co-existing, each of which, adding its own peculiar symptoms to the case, may more or less effectually mask its companions. Thus, for instance, the head of the bone may be obviously dislocated, and yet the characteristic rigidity of the dislocation be absent owing to fracture of the neck, or all attempts at accuracy of diagnosis may be frustrated by the enormous effusion of blood that so frequently accompanies any injury in this region.

The difficulty of accurate diagnosis in such cases is well illustrated by the following case:—A patient of mine in Jervis Street Hospital, with the historic name of John Morley, was sent me from Ballaghareen, in the County Mayo. He had apparently an old-standing dislocation of the head of the humerus beneath the coracoid process, but on

examining the shoulder carefully one could find under the acromion a mass of bony consistence and about the size of a pigeon's egg, or a little larger. He said, moreover, that the dislocation had been frequently reduced, but always recurred. Now I know I am quite sure of your sympathy when I tell you that I made a very bad error in this case, and one not due to ignorance alone, but rather to the influence of the dogmatic teaching I received in my youth. I was convinced that I had to deal with the very rare condition described by the late R. W. Smith, in which, superimposed on a dislocation, is a fracture of the great tuberosity. It is some consolation to me now, in moments of reflection, to know that on that occasion, at least, I erred in the very best company, for my friend Professor Bennett, whose authority in all injuries to bones no one here will question, kindly examined the case for me and came to the same conclusion as I did.

I operated on the man and discovered that there was no fracture whatever; what I mistook for the detached tuberosity was a calcareous mass in the subdeltoid bursa. Now this is a very interesting case to me, and it is rendered still more so by the thought that even had the X-ray method been available then it could not have helped us much, as the mass of lime salts would have thrown as deep a shadow as bone. This is a condition that I have never seen described, and, of course, is not included in any of the numerous classifications of injury in this region.

Having, perhaps, at too much length discussed the question of diagnosis, let me take up that of treatment. Having satisfied ourselves that we are dealing with a case of fracture of the neck of the humerus complicated with a dislocation, how should we treat it? My answer to this question is: by immediate operation in the majority of cases. I am convinced that no other method gives promise of an equally good result.

What are the other methods, the traditional ones, if I may call them so? First, to do nothing whatever to the fracture but begin to set up passive motion as soon as possible, so as to establish a false joint between the head, which is pretty certain to become fixed to the scapula, and the upper end of the shaft. I need not waste your time by discussing this method, which is in itself a confession of impotence and does nothing to relieve

the pain and œdema due to pressure of the displaced head on nerves and veins. Secondly, to endeavour to effect reduction of the displaced head and then to treat the fracture.

Every one who has given any thought to the pathology of this condition will find it hard indeed to believe that such reduction is ever possible. The head cannot be grasped, it can only be pushed with the finger tips, it cannot be rotated. Traction cannot be made on the shaft, lest the few untorn fibres of periosteum be severed and the vitality of the head endangered. Even should the head be forced back into place, one cannot be sure that it is properly applied to the broken end of the shaft. It may be turned topsy-turvy, or it may be implanted in the shaft far below its normal position. However theoretically attractive such a method may be, it is, I think, of little practical value. Thirdly, comes resection of the head. As this involves a cutting operation, it is not less serious than the plan I have recommended of reduction after incision and exposure of the parts. In recent cases it would only be justified after the former had been tried and failure resulted. In old-standing cases it is practically the only method open to us. The pain and œdema of the arm and hand cannot be relieved until the bony mass of the head is taken off the vein and brachial plexus. The result of resection all the same is, I believe, functionally better than that given by a false joint with the head ankylosed to the scapula.

On summing up these arguments and weighing them carefully, I think one is forced to the conclusion that when a dislocation of the head of the humerus complicated by fracture of the neck occurs in a young, healthy man, especially one who has to live by manual labour, the surgeon ought to make up his mind to operate on it at the earliest possible date. This being granted let us consider what method of operating is likely to prove the most useful for our purpose. My own experience is too limited to make dogmatic assertions on the subject, but I am pretty well convinced by such experience as I have had on the subject, that the anterior incision alone is not sufficient for us. It does not give a sufficiently free exposure of the parts to enable the necessary manipulations to be carried out freely and quickly. My belief is that the cap formed by the deltoid muscle must be raised before one can get proper access to the

parts. This can be done either as suggested by Professor Kocher, by detaching the spine of the scapula along its entire length from the rest of the bone, disarticulating the acromial end from the clavicle and throwing the entire mass down, or by detaching the deltoid insertion from the shaft of the humerus and raising the muscle. This latter seems to me the preferable method, as it does not involve such a serious addition to an already grave injury as Professor Kocher's method, nor does it necessitate the subsequent wiring into position of the detached scapular spine. My plan would be as follows:—An incision starting from the clavicle above the coracoid process would follow the line of separation between the deltoid and great pectoral, as far as the insertion of the former. The periosteum here would have to be raised corresponding to the line of attachment of the muscle, or even a small flap of the bone taken with it. The incision would then follow the hinder edge of the deltoid as high as the point at which the circumflex nerve enters it. The muscle could then be turned up very fully, and the seat of injury thoroughly exposed. By means either of a screw inserted in it, or McGill's hook, the dislocated segment could, I believe, be brought into its normal position, and then wired on to the shaft. Such an operation can only be a success if performed very soon after the injury occurs. If any time be allowed to elapse, the shrinkage which takes place prevents either reduction of the dislocation or perfect coaptation of the segments.

What are the objections to this comparatively simple and easily understood operation? That it makes a grave injury still graver, and puts in danger a life that was not previously imperilled. Surely, to say that such an operation imperils a patient's life is really a confession of inability to carry out an operation in accordance with modern principles. The operation should not be dangerous, and never will be in the hands of a careful, competent, and cleanly surgeon. An operator who feels he cannot guarantee his work against infection in such a case should not operate at all; he should seek some other outlet for his energies more suitable to his capacity.

Again, say opponents of reform, this injury does not threaten a man's life, therefore we are not justified in putting him to any risk whatever. Is danger to a man's life the greatest danger he can

face, compared to which all others sink into insignificance? Is the poverty, due to inability to labour with his hands, to which a man the victim of such an injury as that under review is inevitably doomed, of no importance? Are the persistent shooting pains, the paralysis and œdema of hand and arm of no consequence?

My object to night will have been achieved if I succeed in impressing upon my hearers how open to reproach at present is the attitude of most Irish surgeons in hesitating to apply to the treatment of injured bones and joints the same boldness and scientific skill which they unhesitatingly apply to the head, thorax, and abdomen. I feel assured it will not be many years before we will see as great a revolution in the treatment of such cases as we have witnessed during the last twenty years in the treatment of penetrating wounds and other injuries of the abdomen, and those who come after us will look back with contemptuous pity on the unreasoning fatuity of the men of our day, who, with the light of modern knowledge shining brightly on their path, were so fettered by routine and the teaching of tradition that they shrank timidly from travelling that path, and left to another generation the glory of reaping the harvest which it should have been their pride and privilege to garner. Surely no one who has ever seen a working man with an old-standing dislocation and fracture of the upper end of the humerus will hesitate for a moment in urging such a one to face any and every risk to be relieved of his incapacity. Such cases are not commonly seen in the general hospitals, but they can be seen in abundance in the workhouse hospitals, where these poor creatures are compelled, though often yet in the very prime of life, to spend the remainder of their days, through inability to earn their own living by the labour of their hands. That such a state of affairs should still exist is a reproach we should labour diligently to remove

Acute Vomiting.—Louis Kolipinski has used lager beer successfully to stop acute vomiting such as occurs in abdominal and pelvic diseases in females, nervous disorders, and in acute infections. Singularly good results have been obtained from small doses—a wineglass of beer, repeated in half an hour if necessary. The dark beer is preferable.

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A CLINICAL LECTURE ON CONGENITAL AND TERTIARY SYPHILIS OF THE AIR PASSAGES.

Delivered at St. Bartholomew's Hospital,
December 16th, 1901.

By **ANTHONY BOWLBY, C.M.G., F.R.C.S.**

You will remember, gentlemen, that at my last lecture I was speaking to you about primary and secondary syphilis of the nasal and air passages in general. I want now to turn to the affections of these regions in congenital syphilis and in tertiary syphilis. You know that congenital syphilis differs in many ways from acquired syphilis. It manifests itself in some cases very early in life, but in other cases it is not evidenced until after the lapse of months or years. In the case of syphilis of the nasal passages the disease manifests itself more early in life than in any other place almost. You know that the common name for this condition is "the snuffles." If you were to examine the nasal membrane in a child who has what is called the snuffles, you would find there was a considerable occlusion of the nares by the accumulation of crusts; and if you remove those crusts you find in many cases nothing more than a general catarrhal swelling of the mucous membrane, with no ulceration or destruction of it whatever. If, however, you examined a little deeper you would often find in the submucous tissue that there was a gummatous deposit and a thickening of the periosteum. In a great many cases there is nothing more than this general thickening; but in a small proportion of patients, again, upon this condition there supervenes ulceration, so that, even in infantile life, in a few months there may be caries, and sometimes even extensive necrosis. But you must remember that these conditions are rare, and that the usual manifestations are those which I mentioned at first: a seeming catarrh, a swelling of the mucous membrane, and the formation of crusts. Usually there is some thickening, but seldom more than that. Occasionally there is open ulceration and caries, and even extensive necrosis in the early months of life. In a great many cases of congenital syphilis there is no evidence of nasal syphilis in infantile life, and even when there is evidence a child who is treated often recovers, so that after-

wards the only thing which remains to show for the disease is that the nose assumes a curious shape, the end being lumpy, tilted up, and swollen, and altogether out of proportion to the size of the nose as judged by the nasal bones and the lateral masses formed by the superior maxillary bones. The consequence is that the nose in many children, without being extensively destroyed or deformed, is of a characteristic shape, with a slightly depressed bridge, and rather a "blob" at its end. In other cases there is left a real impairment of growth of the bone, so that the bridge of the nose may never properly be developed at all, and the child may grow up with a sunken nose, though there has not been definite external evidence of disease as shown by destruction of bone. It does not therefore necessarily follow that because a child has no evident perforation of the septum of the nose and no necrosis, his nose will not be ultimately of an altered shape. It may be of an altered shape, though neither of these conditions has obtained. In other cases again, unfortunately, as I have mentioned to you, there are extensive caries and necrosis, and the whole shape of the nose is permanently altered, so that the patient may be said practically to have very little nose at all, as far as prominence on the face is concerned.

Later in life, when the time for infantile syphilis has passed by, there may be a condition such as I show you in this drawing. This is the face of a child of eight, which presents the condition known as syphilitic lupus, or a lupoid syphilide. You know that in describing syphilitic eruptions terms are employed which indicate more simple eruptions. For instance, we talk of a syphilitic psoriasis, which is not psoriasis at all; what is meant is merely that it is a syphilitic eruption which has the appearance of psoriasis. We talk of syphilitic acne, and that, again, is a syphilitic eruption which bears a resemblance to acne. In the same way we talk of syphilitic lupus, which has nothing to do with lupus; it is syphilis having the appearance of lupus. This condition, which may be called syphilitic lupus, or a lupoid syphilide, or tertiary syphilitic ulceration of the nose, is a disease which may supervene at any time after the period of the second dentition. It is not very common, but it is sufficiently so to be well recognised, and therefore it is a condition which you have to differentiate from typical tubercular lupus. It differs from

it chiefly in the rapidity with which it extends, and the amount of destruction of tissue which is involved, as well as in the way in which it passes far beyond the ordinary limits of lupus if it is allowed to go untreated. You will see in this next picture still further evidence of the destruction which may result from the extension of the disease. This is the face of a child of nine, whose teeth present the characteristic notches which are supposed to be due to syphilis. Evidently the child had congenital syphilis. In this child's nose is seen, at a later stage, what was seen in the first picture—an extensive ulceration of the skin, a wide opening of the whole of the cavities of the nose, and the destruction of a great part of the ala on the left side. That illustrates a much more extensive destruction in depth of tissue than you get with lupus until the lupus has been going on for years, and usually not then, unless it has extended over a very large surface of the face. This, then, is evidence of nasal syphilis at a stage of life later than infancy. At the same time that this happens, and rarely before, there may be ulceration inside the nose, and there may be ulceration of the pharynx. The larynx, fortunately, is rarely affected in congenital syphilis, although I shall tell you presently that in tertiary syphilis it is. But the tongue is affected in congenital syphilis in a good many cases. Here is a good picture of a child, a boy *æt.* 7, who had been under treatment one and a half years, and here you get the typical conditions of tertiary syphilis of the tongue. So you see that there may be not only affections of the nose in children, but there may be affections of the pharynx with ulceration, and affections of the tongue of the same nature as those with which you are familiar in cases of acquired syphilis in the tertiary period. As far as these patients are concerned, you may say in general terms that the diagnosis is tolerably simple. There is not much else that looks like an ulcerative syphilide of the nose; there is nothing which closely resembles this condition of the tongue, and therefore, without wasting too much time upon the matter, as it is a subject which one has hardly time to discuss now, it is enough to say that the diagnosis is easy, and that the treatment to be adopted is that for syphilis in general, not only by means of iodide of potassium, but also by mercury. Both of these are useful, and if ad-

ministered in reasonable doses and continued for a sufficient time, the results in these cases of syphilis of children, as apart from the cases of infancy, are thoroughly satisfactory.

I need hardly remind you that the general treatment of syphilis in infants is very unsatisfactory. A large number of such patients are sure to die very young, whatever you do for them. The evidence which you may detect of the disease in the nose is not the only evidence which will guide you in the diagnosis, but it will enable you to realise that probably the child has syphilitic disease going on elsewhere in its body; you will be able to see that its viscera are very likely unsound, and that apart from the condition of its nose, it is likely to die. Moreover, the trouble in the nose causes some interference with the vitality of the child, and often makes it very difficult for it to suck its food. Thus the chance of recovery of a sick child is very materially prejudiced, not so much from the amount of destruction of tissue as on account of the difficulty which it has in taking its nourishment.

We will now leave hereditary syphilis, though there are many more things which might be said about it, and turn to tertiary syphilis, and here again we begin with the nose. Tertiary syphilis of the nose may attack either the external surface of the nose or the inside of that organ. Let us say a few words about the external surface first, and dismiss the subject. A person with acquired syphilis may have such a condition of the nose as I pointed out to you as occurring in childhood, that is to say, a so-called ulcerative syphilide, or syphilitic lupus. This, then, is a condition which may occur both in congenital and in acquired syphilis, and you may expect to find patients with tertiary syphilis who have destruction of the ala of the nose, or of a considerable portion of its tip.

Next, with regard to the inside of the nose. Here, of course, you have to do with conditions which are very much more serious, and this chiefly because they are so latent, and therefore liable to be overlooked. If you examine the inside of the nose in cases of tertiary syphilis, at the beginning of some syphilitic affection of the inside of the nares, you may find, either on the outer side of the nostril, on the turbinate bodies, or on the septum (the latter being the more common), a swelling. No ulceration precedes this as a rule. That

swelling is due to a gummatous deposit in the periosteum, and sometimes in the substance of the turbinate bodies. All that the patient complains of is some difficulty in breathing through the nose. The swelling is soft, and if the condition is on the outer side of the nose you are liable to say that it is hypertrophic rhinitis, and indeed it looks like it. But if you examine further you will find there is a much more substantial swelling; it has not the oedema which you associate with enlargement of the turbinate bodies. This swelling is different to that; it is much more thick and substantial, is often not limited to the turbinate body, and does not disappear when you put cocaine into the nose. So you have something which looks like hypertrophic rhinitis, and which may be mistaken for it. At that stage, if you recognise the disease and treat it, you may get your patient well without trouble occurring inside the nose beyond the temporary blocking and consequent difficulty in breathing. The whole of this swelling may be entirely removed by the administration of iodide of potassium and mercury, and the patient may completely recover without having a sore place inside the nose at all. You are less likely to make the mistake of not recognising this early condition when it affects the septum, because swellings of the septum are not very common apart from syphilis. They occur in tubercle, but generally the patients we are speaking of are older than patients who have tubercular swellings of the septum. Then there is new growth, but the cases are not like that. In cases of syphilitic disease of the septum there is generally a thickening and evident swelling of one side. Often, however, both sides of the septum are involved, so that you may have enlargement of the septum as a whole. If you recognise this condition you can often prevent the patient from having anything more the matter with his nose, because at this stage the condition is amenable to treatment. If you wait, or if the patient does not see you soon enough, what happens? What happens on the outer side, in the condition which resembles hypertrophic rhinitis? What happens in the septum where there is not yet any ulceration? These swellings will ultimately break down, they will ulcerate, and they will leave an open place which discharges purulent and blood-stained material, which is the débris of the tissues which have been destroyed. How much tissue has been destroyed

varies immensely in different cases. In one case when the gumma bursts and discharges its contents there is found to be an ulcer, at the bottom of which the bone may not be evidently exposed, or the bone is perhaps exposed over only a very small area; and that ulcer heals up. Or it may be that there is an ulcer, at the bottom of which carious bone is extensively exposed, and this will take a long time to heal. These conditions may extend, the ulceration may progress, more bone may become carious, and ultimately the whole inside of the nose may become widely and extensively ulcerated, with perforation of the septum, and necrosis of other bones. At any rate, the state of the patient after a time is that a great deal of the nose is destroyed from within. Whilst these changes are taking place, in not a few cases there is no necessary alteration in the external shape of the nose, even although there may be extensive destruction of the septum. You will be able to see patients in the throat department in whom there is a large hole in the septum of the nose, and yet the bridge has not fallen in. I want you to remember that, because it seems to be the opinion of many people that as soon as the septum of the nose is destroyed the nose falls in. But the nose does not fall in simply because of that. The nose is supported laterally by the nasal bones and the superior maxillary bones, and these do not necessarily fall in. These make the bridge. The nose does not depend merely on the support which it receives from the septum. Therefore in many of these cases you can see destruction of bone, and yet, though there is a complete breaking open of the cavities of the nose, so that they form one large cavity, there is not necessarily any material deformity. In other cases, unfortunately, there is much material deformity, and such large portions of the nose may be destroyed that either the bridge of the nose may sink in and leave the fleshy part of the nose projecting as a shapeless mass, or ulceration may extend to the tip of the nose and all the soft parts of the organ may be destroyed. In some cases both of these conditions go together, that is to say, a destruction of the soft parts and of the bony framework of the nose.

With regard to the differential diagnosis, when it is a case of ulceration there is, as a rule, very little difficulty in diagnosing the condition; but you have to keep in mind that there is a form of

ulceration of the septum or the nose which is not due to syphilis, and which is called "atrophic ulcer" of the septum of the nose. It occurs practically only in women, and almost entirely in women over forty years of age. It has no definitely recognised cause. Tubercle also may perforate the nose, but that almost always occurs in quite young subjects. Perforation of the septum of the nose by syphilis is the most common. Atrophic ulceration of the nose, let me say at once, is a very slowly extending form of ulceration, which may very likely take a year or two to perforate the septum. It occurs immediately behind the tip of the nose, and as it occurs in that place it usually perforates the cartilage and not the bone. Ultimately it gradually cicatrises at its edges, leaving, however, a permanent hole through the septum of the nose. That is a curious condition, the pathology of which we do not understand. But you may say definitely it is so unlike the course of syphilis that you are not at all likely to mistake the one for the other.

As to symptoms, I have already told you that at the beginning the only thing which the patient will complain of, before there is ulceration, is some obstruction to the passage of air through the nose. You can easily understand that; afterwards, when there is ulceration, the patient complains of discharge from the nose, and it is to be noted that in many cases the discharge is characteristically blood-stained. If you find a discharge from the nose which is practically always blood-stained, the case is most likely to be one of tertiary syphilitic ulceration. If, however, the patient be a child, and there is a blood-stained discharge from the nose, remember that the commonest cause for that is undoubtedly the presence of a foreign body in the nose, and I need hardly say this is something altogether different from what we are considering. We are speaking about tertiary syphilis now, in which the patients are adults, and if there be a profuse blood-stained discharge from the nose, that is almost always an indication of tertiary syphilis. Other discharges from the nose are seldom regularly blood-stained. Later on the patient may complain of loss of the sense of smell, which is due to the destruction of the mucous membrane, and of the terminal filaments of the olfactory nerve. Later on still the patient may have *ozæna*, of which, however, he will not complain so much as will his friends. *Ozæna* is a characteristic fetid

odour which may occur from other causes than syphilis, but it is one of the great troubles of tertiary syphilis, when ulceration has extended and destroyed much of the bone, and is associated with caries or necrosis.

What you will be able to do for your patient will greatly depend upon the state in which you see him. In all these cases the ordinary antisyphilitic remedies should be employed, and always remember to employ mercury as well as iodide of potassium. I am no advocate of giving iodide of potassium and withholding the mercury. You have also to carry out measures for local cleanliness, such as the washing out of the nose with salt and water, and afterwards the introduction of an antiseptic powder. In the throat department we have been using lozenges and borax. The first of these is practically iodoform under another name and in another form. It has about the same proportion of iodine, which it liberates in contact with living tissues, and thus acts as a disinfectant. We use it instead of iodol because it is cheaper and because it is insufflated much more easily. After having used other preparations we have come to the conclusion that on the whole lozenges is better than most things. I always use it with borax, because borax liquefies mucus and promotes a more watery discharge from the nose, and seems to prevent to some extent the formation of the thick crusts and scabs. I am not particular as to the exact form of antiseptic which you may use. The essential thing is that you should wash the nose out to get rid of the crusts and scabs, and afterwards that the patient should use some antiseptic powder to blow into the nose. Most of these cases yield to treatment, but a few of them do not.

And now let me point out one of the results of syphilitic ulceration of the nose when the ulceration has ceased—cicatrisation ensues. In the case which is illustrated in the picture I show you cicatrisation has ensued to an extent which is very rare; nearly the whole of the nares are completely occluded. There is an absolute stoppage of the nostrils by scar tissue, so that the patient has no breathing room at all. This is a complication which you have to bear in mind, but it does not often happen. When there is complete occlusion of the nares as a result of ulceration of a syphilitic nature, the cases are most difficult to treat. The treatment, indeed, is almost sufficient

to occupy a lecture by itself. It is very difficult to restore a passage or channel in any part of the body which has been closed by a cicatrix, and you have, in such patients, to perform some plastic operation.

Let us now pass to the other end of the nasal passages, namely, the pharynx. There is not so much to be said about the pharynx as about the nose in tertiary syphilis. But the back of the pharynx may be ulcerated, and so may the roof of the mouth and the soft palate. Let me remind you that the roof of the mouth is the floor of the nose; what is the ceiling of one story is the floor of another. The result is that ulcerations inside the nose may result in perforation of the palate, and you may feel, before there is perforation, the same swelling which I have mentioned as occurring inside the nose before ulceration commences there. If you are examining a patient with syphilis, and if you find, after having satisfied yourself of the evidence of syphilis inside the nose, that there is a bulging on the upper part of the hard palate, you may be sure that if you do not get that patient quickly under the influence of treatment there will be perforation inside the mouth. At a later stage the palate may be ulcerated, and so may the pillars of the fauces and the pharynx. These ulcerations you may see either at the time that they are recent and open, or you may see them later, when the process of cicatrisation is going on. The picture which I have shown you of occlusion of the anterior nares represents the similar condition which may occur after ulceration in the pharynx. A little time back a patient came here quite unable to breathe through the nose. The examination of the anterior nares showed nothing, but, on opening the mouth, you could see that the palate was drawn upwards, and was adherent to the pharynx, that the pillars of the fauces were dragged upon, and that there was only a small hole left through which one could pass a fine catheter into the nasopharynx. The whole of the rest of this space was shut off by an extensive scar, the result of tertiary syphilitic ulceration of this region, which had destroyed the uvula and the pillars of the fauces.

And now, in conclusion, a few words about tertiary syphilitic ulceration a little lower down, namely, in the larynx.

With regard to the larynx, there is not so much to be said, but what is to be said is of very great

importance. In many cases a patient who has tertiary syphilis has a hoarse, harsh voice. If you examine him you will find either swelling or ulceration to account for it. The swelling may be an ordinary gummatous swelling, entirely analogous to that which I described as taking place in the septum of the nose or the turbinate bones, and you may find on one side of the larynx a lump which, we will say, is above the vocal cord, for the ventricular band is a very common place for this condition, and so is the ary-epiglottic fold. As that swelling increases it infiltrates the muscles, and although the cord itself is free and may appear normal, the movements of the cord may be hampered, and the patient, as a result, talks with difficulty. If you see the same patient later on, you may notice that the swelling has disappeared and an ulcer has come in its place—the gumma has broken down. Here, in exactly the same way as in the nose, there may be superficial ulceration or deep destruction of the muscles in the larynx, of the ligaments, or of the crico-arytænoid joint, and as a consequence there may be a permanent alteration in the movements of the cord, owing to the destruction of the muscles, the formation of fibrous tissue, and perhaps to ankylosis of the crico-arytænoid joint. Then, in exactly the same way as gummatous infiltration in the nose extends to the periosteum, so it may extend to the perichondrium and the cartilages, although, fortunately, this is more rare. It may thus give rise to perichondritis, with effusion underneath the perichondrium, entirely analogous to periostitis, so that sometimes one side of the larynx may be blocked by a large swelling which completely occludes that side of the larynx and quite hides all the normal tissues. All that you can see in such a case is a large, deep-seated swelling lifting up everything, and making the whole of the anatomy of the larynx so completely altered that you can hardly recognise it.

Let us now go a step further. Ulceration may result in destruction of cartilage. We have got as far as perichondritis, and the ulceration may go on to ulceration of the cartilage. Of all cartilages which are likely to be destroyed the chief is the epiglottis, which is destroyed more often than is any other in cases of tertiary syphilis. Here is a picture which shows very widespread infiltration and ulceration inside the larynx, and here is

another, from a man who was forty years of age, in whom there has been very extensive destruction and ulceration of the essential parts of the larynx, including some parts of the vocal cords. I have already said that hoarseness is the characteristic condition early in these cases; and the other thing to be noticed is that this condition of ulceration and swelling in the larynx in syphilis is almost painless. It thus differs materially from many, though not from all cases of tubercle, for many cases of tubercular laryngitis are very painful. Syphilitic laryngitis hardly ever causes pain, even if it is very advanced. Later on, not only may the voice be materially altered, as the parts are infiltrated, the muscles destroyed, and the edges of the cord ulcerated, but, on account of the amount of swelling, there is sometimes considerable dyspnoea, and if treatment is not applied sufficiently quickly you may have to do a tracheotomy. You may find that a patient who has not had dyspnoea will, on a certain day, within a few hours of your seeing him, develop severe dyspnoea. The explanation is that in a case where there has been already an open ulcer of the larynx, the patient may, by exposure to cold or wet, have an ordinary catarrh, and where there is already much swelling of the larynx the swelling which supervenes from the catarrh is sufficient to turn the balance and make it difficult for him to get a sufficient quantity of air down into the lungs at all.

Later on, as ulceration ceases and cicatrization ensues, the larynx may be contracted in the process, and so there may be stenosis. The result may be that after the ulceration has ceased the dyspnoea may be much worse than it was during the active process of ulceration. It will be serious in proportion to the amount of tissue which has been destroyed and to the fixity of the parts which are left; so there is no hard and fast rule about these cases. Fortunately a large majority of the patients who have syphilitic ulceration of the larynx recover without any permanent dyspnoea, though with altered voice and permanent hoarseness, conditions due to the damage which has been done to the vocal cords and to their fixity, which is irrecoverable. But most of them have not any permanent dyspnoea, though some of them may have some shortness of breath on exertion now and again. You may, however, meet with cicatrization, analogous to that which I showed you in

the case of the nose, which may result in a gradual drawing in of the orifice of the glottis, so that dyspnoea may be serious, and this dyspnoea you may have to treat by the use of a permanent tracheotomy tube. The dyspnoea, which is the result of the swelling occurring while the syphilitic processes are in progress, you may also have to treat by tracheotomy, but in the majority of such cases, where the dyspnoea is due to the inflammatory swelling, the use of a tube will only be temporary.

You will see, then, that in general terms syphilis, as it affects the nose or the pharynx or the larynx, begins, in a large number of cases, when it is in the tertiary period, in a swelling. If you recognise it when there is only swelling you have recognised it in time to prevent very much damage in a large number of cases. In the second stage this swelling is succeeded by ulceration, with destruction of tissue to a very varying extent. Thirdly, there ensues cicatrisation, and that may result either in the natural healing of the ulcerated parts, leaving no other damage, or it may result in stenosis, so that there may be narrowing of the nares, or even occlusion and stenosis of the larynx, causing more or less serious dyspnoea.

Demodex Folliculorum Disease.—Several years ago Dr. C. W. Allen showed a woman at the New York Dermatological Society with a peculiar eruption on the face, resembling more than anything else minute papules of *Molluscum contagiosum*, and this was thought to be the disease present. He called attention to the number of *Demodex folliculorum* observed in a state of vigorous activity, and stated his belief that this large parasite must be of more ætiological importance than text-books had ever accorded it. Since then he has taught that the demodex was probably something more than a passing tenant of the follicles of the skin. Dubreuilh reports a case of pigmentation of the face showing prominent hair follicles from which a minute horny spine projected. Three months later, after treatment, the condition persisted, and so did the parasites. Severe skin affections are recognised in the dog and pig as due to a very similar demodex, and it really seems time to grant to that variety which favours the human race some reason for existing.—*The Post Graduate*, Jan., 1902.

A CLINICAL LECTURE

ON

A CASE OF ACUTE MYELITIS.

Delivered at Sheffield Royal Hospital,
January 7th, 1902.

By ARTHUR HALL, M.A., M.B. Cantab.,
M.R.C.P.,

Physician, Sheffield Royal Hospital; Professor of
Pathology, and Joint Lecturer on Clinical Medicine,
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GENTLEMEN,—The patient about whom I am going to speak to-day was in the Royal Hospital a few months ago. As he is now convalescent and able to get about, although not yet completely recovered, I have brought him up to-day in order that we may discuss his case. The history of his illness is full of interest, and for the excellent notes I am indebted to the house physician, Dr. J. H. McKee, and to one of my late clinical clerks, Mr. P. Drabble.

The patient was a gardener, æt. 34, and was admitted to the Sheffield Royal Hospital on September 13th, 1901, complaining of loss of the use of his legs, also loss of control over his sphincters. He stated that he was quite well till August 30th, when he began to have diarrhoea, which lasted a week and then got better. After the diarrhoea vomiting set in on September 6th, the attacks of vomiting coming on chiefly in the night. With the vomiting he began to lose the use of his legs; he was able, however, to come downstairs the first three or four days of his vomiting and weakness, but after this the power in his legs went altogether and he was obliged to remain in bed. The change in his legs began as follows:—First a feeling of weakness with tingling and numbness, then his legs felt cold and he lost the power in them. He thinks the left leg gave way first. He is married, but has no children living; one died young of marasmus. His father died of heart disease, and his mother of a cause unknown. He does outdoor work, lives in a good healthy neighbourhood, and gets plenty of good food; he takes a little beer at supper but not at other times; he has never lived abroad, and there is no previous history of venereal disease; he does not remember having been exposed to any chill previous to his illness; he has not had any pain anywhere with the exception of a feeling of soreness on his buttocks

due to a bed sore. When seen on admission he lay on his right or left side with the knees slightly flexed; the hips and shoulders were not kept in the same plane, for while the former remained fixed the latter were capable of being moved about; he could move his feet and toes slightly, also his legs a little; he could flex and extend his feet slightly, the right better than the left. His intelligence was only moderate, but his general state of development and nutrition was good. The bowels acted at intervals and were not under the patient's control; the circulatory and respiratory systems were normal; there was almost complete loss of power of the pelvis and lower limbs—all he could do was to move the latter just perceptibly; the extensors were not more affected than the flexors; the left foot was slightly more affected than the right; there was no marked wasting; sensation was much impaired but not lost absolutely; it was very difficult to make out accurately, and his statements were very contradictory, but he could distinguish heat, cold, touch, and pain vaguely. The upper limit of the sensory change was very hard to define; both knee-jerks were increased, the left more than the right; the plantar reflexes were present on both sides but gave him a sensation of discomfort. On the buttocks there was a bright red circle of inflammation, covered with small blisters about one inch broad, caused by his having placed a chamber-pot under the buttocks whilst in bed on account of his diarrhoea. He had kept this under him for some hours and the bed sore corresponded exactly to the rim of the po. This, in the upper half, ulcerated deeply later, causing a serious bed sore. There were no ocular symptoms; the fundus of each eye was normal. There was complete loss of control over the sphincters of bladder and rectum, each of the acts being perfectly carried out at intervals without his knowledge. There was a little pus in the urine, but it was only very slight and the reaction was acid. He ate and slept well. Temperature 101.8° in the evening and normal in the morning.

In reviewing his condition on September 13th we find that the patient had commenced on August 30th with diarrhoea, that after a few days vomiting occurred also, and about the same time that various nervous symptoms developed, limited entirely to the lower part of the body and the lower extremities, causing a so-called *paraplegia*.

Together with these the patient had, on admission, a raised temperature, which lasted for about a week. On further analysing the evidences of nervous disease we find—

1. Almost complete paralysis of the muscles of the pelvis and legs on both sides.
2. Impaired sensation over the corresponding parts of the skin.
3. Absolute loss of control over the sphincters of the bladder and rectum with perfect performance of the acts.
4. Slight exaggeration of myotatic irritability, the left more than the right.
5. Persistence of cutaneous reflexes.
6. No wasting of muscles.
7. Marked inflammation over a semicircular area of curious shape on the buttocks, following the sitting upon a chamber-pot during the night.

Let us take these points *seriatim* and see where they help us to a diagnosis.

1. Paralysis almost complete of all the muscles of the pelvis and legs might mean (*a*) Disease in muscles themselves; (*b*) in nerve-trunks; (*c*) in cell-bodies of spinal neurons; (*d*) higher up in nervous system.

Which is it in this case? (*a*) It is *a priori* impossible to say from this evidence alone what part is diseased; it might be any of the above, but we may judge something from the distribution of the paralysis. Thus, as regards the muscles themselves, we can conceive of no kind of affection of the muscles themselves which in the short space of a few hours could render them unable to be contracted, except it were due to some poison circulating in the blood, and, if so, there is no anatomical vascular arrangement by which the affected muscles should be attacked to the exclusion of all the remaining muscles in the body. Besides, organic destruction of muscle tissue is always a slow or chronic process, never acute like this.

(*b*) Is it in the nerve-trunks alone? This is possible, but here again it is improbable, for we know that poisons which act upon the peripheral nerve-trunks, such as alcohol, lead, diphtheria, etc., causing so-called multiple neuritis, with consequent paralysis, almost always select certain nerve-trunks upon which they act first and most readily—why this is so we do not know, but the fact remains. Now these “spheres of action” are sometimes in the legs, sometimes in the arms, sometimes else-

where, but they are not commonly in the form of a simultaneous paraplegia at their commencement.

(c) Is the disease in the cell-bodies of the peripheral neurons, that is, in the interior cornua giant-cells? From the paralysis alone it might be so; or (d) is it higher up in the nervous system? Again from the paralysis alone it might be.

2. The second fact observed is that there is impaired sensation over the corresponding parts of the skin, that is, over the whole of both legs and the lower part of the trunk.

This at once removes the possibility of the disease being in the muscles, as you can readily see, even had we not, for other reasons, thought it unlikely; we may therefore dismiss that at once. It may still, however, be the peripheral nerves that are affected, for they contain both motor and sensory fibres; but, as we have seen just now, that is unlikely from the form of paralysis. If the disease is in the bodies of the peripheral neurons in the anterior cornua of the cord it cannot be limited to them, for you know from your physiology that these have nothing to do with the receiving of centripetal or sensory impressions, and that sensory and motor nerve-fibres separate at the junction of the nerve-trunk with its anterior and posterior root, entering the cord in front and behind respectively. If, then, the lesion is in the spinal cord, it must be involving something more than the anterior horns, and extending from back to front.

3. The next fact on our list is that there is loss of control over the sphincters. Let us see in what way these visceral reflexes are affected. As soon as the man's bladder gets full it empties itself into the bed without his knowledge, the same with his bowel. The reflex action is therefore perfect enough, and so well does the urinary reflex perform its duties that there is hardly any residual urine on catheterisation. It is evident therefore that the lesion does not involve any part of the reflex arcs, not even the centres for micturition and defæcation themselves. Thus the peripheral nerves may be dismissed as the seat of the disease, and the cord, at any rate as far as the top of the lumbar swelling. The lesion must be higher up or brainwards in the cord. How high up is this lesion of the spinal cord? Evidently only so high as that segment of the cord which supplies the motor and sensory nerves to the upper portion of the area of

the body affected. As we saw, this upper margin was not easy to define, but was somewhere about the lower half of the abdomen. We may therefore place the lesion in the lower thoracic region of the cord. And since impulses going up from the periphery or coming down from the brain are stopped at that point the lesion must extend fairly widely across the cord; but all impulses are not absolutely stopped, he can just move his legs slightly, and he can detect sensations very vaguely; therefore the obstruction is incomplete. This, then, is a case of incomplete transverse myelitis affecting the lower thoracic cord. We cannot accurately define its thickness, but we can say definitely that it does not go lower than the highest part of the centre for micturition, that is to say, that the lumbar swelling of grey matter is not affected. Now let us fit in the rest of the symptoms.

4. Slight exaggeration of myotatic irritability—knee-jerks. This is a constant accompaniment of irritating lesions of the upper neurons. In his case it is more marked on the left than on the right, and this is associated with a more marked paralysis on the left. Therefore his lesion is probably more extensive on the left side of the cord.

5. Persistence of cutaneous reflexes. These, like the visceral reflexes, remain, because their local centre in the cord is not involved.

6. Absence of muscular atrophy. Because the lower neurons are not affected.

7. Acute bed sore. The reason for this is at first sight not so evident, but as a matter of experience it is quite characteristic of the disease. It is a very striking thing; I remember that in the next bed to this patient there was a man who had been lying there for months, and who was wasted to the last degree with diabetes, complicated by pulmonary tuberculosis, and yet he had no bed-sore, whilst this patient with myelitis had unfortunately placed a chamber-pot under his buttocks for a few hours a night or two previously, and as a result had a deep semicircular ulcer, which showed no tendency to heal, and at one time threatened to prove fatal to him. As I say, this tendency to acute bedsores wherever there is pressure is a striking feature of this disease, *and it always and only occurs in parts whose nervous supply is below the level of the affection.* It is commonly said that it is

due to the trophic nerves being affected; it has also been suggested that it is merely due to the loss of sensation preventing the patient feeling when he has laid on one part too long, and his inability to move himself if he has. How far each or all of these factors may enter is uncertain, but the fact remains, and a very important one it is. I have said nothing here as to the electrical conditions of the muscles, for the reason that our constant current battery was at the time out of use—a condition which, in my experience of hospital batteries, is more constant than the current.

The patient continued much the same for the next few days, the temperature being up irregularly, but on September 24th it became normal, and the patient seemed slightly better. A few days later (September 26th) it was noted that his speech became thick and unintelligible, and that he had difficulty in swallowing also, that he was very sleepy and drowsy, and that he was quite foolish mentally. He frequently tried to get out of bed, and for some days was quite insane. He also partially lost the use of his arms, and his temperature after a few days (September 30th) again kept going up irregularly.

Meanwhile he was recovering the use of his legs daily. The temperature became normal again on October 3rd, and the other symptoms gradually disappeared. It is difficult to say exactly what happened during this time, but it seems probable that he had a relapse, and that other scattered foci of myelitis in the cervical cord and bulb were formed; they must have been exceedingly small in size, or in such a situation as the bulb they must have proved fatal.

The strange mental condition coming on quite rapidly and passing off with the other symptoms was possibly due to a similar condition in some part of the brain. Such conditions have been described under the name of encephalo-myelitis. Whether this case can be classed with them or no, it is certain that the patient had a recurrence of his myelitis commencing very shortly after the fever of his first attack had subsided, with mental symptoms and excessive sleepiness, that this was followed shortly by symptoms of paralysis affecting the tongue and throat muscles, and that the pyrexia associated with these symptoms of a relapse did not occur until the fifth day of the relapse.

The further history of the case is one of steady progress and uneventful recovery. It was noticeable particularly that the bed sore, which had remained hitherto *in statu quo*, began to heal rapidly as soon as he began to recover power in the legs. On October 7th he was able to walk with assistance in the wards. He is now (January, 1902) able to walk well, and has almost completely recovered in every way.

This case of acute myelitis was not a very severe one, for the lesion was incomplete, that is, it did not completely cut off all the connection between the cord above and below it. Again, it did not involve the grey matter of the lumbar swelling, so that there was not much muscular atrophy or disorganisation of the visceral reflexes. Had it not been for the unfortunate affair with the chamber-pot, the dangers would have been still less. The most serious item in the case was the relapse involving the higher centres, and at one time this threatened to be fatal.

In regard to treatment, there is no disease in which it is possible to do less directly to check the disease, but in which also it is possible to do more to save life than in acute myelitis. Let us realise at once that drugs given in the acute state with the object of acting on the cord are, in the present state of our knowledge, worse than useless. What, then, can be done? You must act as a "temporary cerebrum" to those parts of his system which are cut off from his own cerebral influences, and which, unfortunately, include two of his chief channels of elimination of waste material. Prevent him from being killed by his paralysed lower half. To carry that out is not easy, but it must be attempted. In the first place, if his brain were able to do so it would keep frequently moving him very slightly, so that he did not lie too long in one position. It would not necessarily be a large movement, often only a little wriggle, but yet quite sufficient to change the line of pressure. You must try to imitate this by seeing that he is frequently moved from side to side, or even semi-prone, the oftener the better. A very slight movement each time will suffice. A water bed may also help in this by distributing the pressure. Another very valuable adjunct which was, I believe, first suggested by my friend, Mr. Dale James, of Sheffield, is to paint carefully over the sites of greatest pressure, the ischial tuberosities, sacrum,

hips, backs of heels, etc., with the preparation known as zinc-gelatine, or the modification of it which is used in the skin department here, and which is more effective, namely, calamine gelatine. Properly done with absorbent wool this gives the skin, so to speak, a kind of extra thick epidermis; in fact, its action may be compared to the mode of protection which the skin adopts itself when under pressure, for example, the hoofs on the labourer's hands; and it has this advantage over any other dressing that it has less chance of becoming wrinkled and causing folds. If a bedsore has already formed, it must be antiseptically dressed and the margin widely painted round with calamine gelatine. It will not heal until the patient begins to recover, but it may be prevented from doing harm.

Next the urine must be considered, and whatever is done it is often most difficult to prevent trouble. It is not easy to keep a urinary vessel over the penis, and the pressure it exerts on the thighs is bad. Frequent catheterisation is also harmful, except under very strict antiseptic precautions. In this case the catheter was passed by the house physician night and morning to empty the bladder, which was then washed out with boric lotion; a large sheet of waterproof tissue was slit up and fastened round the root of the penis and testes, and a suitable bed urinal placed *in situ* surrounded with abundance of absorbent wool.

As regards the bowels, protecting the anus thoroughly with tow and wool and frequent attention, together with scrupulous after-cleanliness and occasional enemata, is perhaps the best that can be done.

Any rough handling of the muscles in the shape of massage, or irritation of the surface by application of electrodes, is to be avoided until the acute stage is over.

Acetonuria in Pregnancy.—R. Costa concludes from his researches that in physiological pregnancy acetonuria is in greater amount than in the non-gravid condition. In labour, especially if it be prolonged, acetonuria is increased. In the puerperal state it decreases, although still remaining higher than during pregnancy.—*American Journ. of Obstet.*, Jan., 1902.

A Text-Book of Medicine. (Fagge and Pye-Smith, Vol. I, Fourth Edition).—Messrs. J. and A. Churchill have issued the fourth edition of the well-known book generally spoken of as "Fagge's Medicine." The work is in two volumes, and if the second volume equals the first Dr. Pye-Smith may congratulate himself that he has earned the distinction of having revised and re-written one of the best publications in medical literature that has yet been offered to practitioners and students of medicine. It may perhaps seem strange to first praise the printing of a book, but so much of the reader's comfort depends on this important point that an appreciation of the typographical excellence of this volume must be permitted. The words of Dr. Pye-Smith in the preface, where he defends the practice of giving an historical account of maladies, are assured of a hearty support from all lovers of the best literary methods. The book has such a world-wide reputation that it seems almost superfluous to recommend it. The following passage on the diagnosis of sciatica will serve as an example of the practical and useful character of the teaching to be found in its pages:—"The *diagnosis* of sciatica is seldom difficult to the practitioner who first searches for the numerous other diseases that may cause pain in the thigh and leg. Where the calf is the part mainly affected one must think of thrombosis of the femoral vein, make digital examination of the vessel at the groin, and look for œdema of the ankle. Disease of the hip-joint can be put out of consideration by the fact that neither pressure on the trochanter nor forcing the head of the femur against the acetabulum, nor extreme flexion, gives rise to pain; and disease of the sacro iliac synchondrosis by the fact that no tenderness is elicited by tapping the articulation, or by pressure of the two ilia together. Advanced cases, in which the patient limps in walking and has wasting of the muscles, are very likely to be mistaken for spinal disease; this occurred some years ago in the case of a medical man whose fellow practitioners in his own neighbourhood all felt sure that he had disease of his vertebræ. Even if one is satisfied as to the seat of the pain, one must still search carefully for local causes of irritation. In one instructive case a surgeon, feeling carefully along the course of the nerve, was fortunate enough to detect the presence of a piece of broken needle, the removal of which led at once to the cure of the patient."

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WITH SIR WILLIAM BENNETT IN THE WARDS OF ST. GEORGE'S HOSPITAL.

INTERNAL DERANGEMENT OF THE KNEE-JOINT.
THIS patient affords a typical example of internal derangement of the knee-joint, from recurrent displacement of a semilunar cartilage. It is a very interesting case, because we have been fortunate enough to get with the patient a very characteristic history. He was in the hospital five years ago under Mr. Pick. At that time, as there had been very precise symptoms of displacement of the right internal semilunar cartilage, the man having had five successive attacks, and the cartilage being apparently loose, Mr. Pick operated. The cartilage was found much detached, and was stitched to the head of the tibia. At that time the value of the operation of suturing a loose semilunar cartilage had not been ascertained. Some people thought it was a very valuable proceeding; others, on the other hand, thought very little of it. Of course the relative values of treatments can only be determined by experience, and it is now admitted by the majority of rational surgeons that the operation of merely stitching a loose semilunar cartilage is, for curative purposes, practically useless. This man now comes with the history that five months after the operation he had another attack exactly like those which occurred before it. In jumping across a ditch recently he alighted roughly on his leg and "twisted" the knee, upon which the locking of the joint and other symptoms with which he was familiar occurred. In all these cases the attack is brought on by a *twisting* of the limb; it is never a fair to-and-fro movement which brings about the displacement; there is always a rotation of the tibia on the femur. The first recurrence after the operation was five months subsequent to the suturing five years ago. The patient has therefore been going on for something like four and a half

years having these constant attacks of displacement of the cartilage, which means that he gets sudden attacks of pain, the joint becoming fixed in a slightly flexed position, and until he, either by his own effort, puts the leg straight, or a friend does it for him, he is helpless. Each of these attacks is followed by some effusion into the joint, so that the patient is becoming more and more incapacitated for work. There is an advantage in this case in knowing that, as the joint has been investigated, it is one of displacement of a semilunar cartilage. The importance of this point lies in the fact that it is extremely difficult to diagnose with certainty the causes of internal derangement of the knee-joint; it is difficult to know whether the trouble from which the patient suffers is due to displacement of a semilunar cartilage, to the getting between the bone-ends of an hypertrophied synovial fringe, or, what is more common, to the hypertrophy of the folds of synovial membrane which lie behind the lower end of the patella and its ligament. It is stated that in this case there can be felt at the time of the attacks a small lump sticking out from between the bones. We know that is the case, because Mr. Mummery, my house-surgeon, who had an opportunity of seeing the patient about six or seven months ago, when he came to the hospital on account of an attack, found a distinct lump projecting from between the tibia and the femur, on the inner side. This symptom is quite unusual in the ordinary way in cases of displacement of a semilunar cartilage. As a rule, displacements of semilunar cartilages take place into the joint, and not out of it. It is from the fact that the displacements generally take place into the joint that the symptoms resulting from the condition are so very acute. If the displacement were entirely outwards there need be no jamming of the loose body between the bones, and not necessarily any locking of the joint. If the displacement occurs inwards, as it almost always does, at all events to some extent, the foreign body—for such it becomes when it is displaced—gets between the bones and locks the joint. As a rule, therefore, in a semilunar cartilage displacement you feel very little that is abnormal. You may feel a deficiency, but not enough always to base any definite conclusion upon. On the other hand, if an hypertrophied synovial fringe, or especially if an hypertrophied fold behind the ligamentum patellæ be caught between the bones,

a rounded lump is generally found projecting from between them. You may naturally ask, What does it matter whether the symptoms are due to semilunar cartilage displacement or whether they are caused by the catching of some odd synovial fold, or something of that sort, between the bones? As a rule it certainly does not much matter, but in some cases it may affect the choice of the incision. In the present case we know the semilunar cartilage is at fault, and that we have to recommend the treatment which will, as far as we know, effect a cure. The only treatment which we have to rely upon here is the removal of the faulty semilunar cartilage, which is not a very serious operation. But I wish you to bear in mind a very important point in relation to the cure of these cases; it is this: After the removal of a semilunar cartilage it does not at all follow that the patient will immediately get quite right. In a case of this sort you will find that the joint affected is always looser and more “wobbly” than the one on the other side, in consequence of the recurrent attacks of joint irritation, generally followed by some effusion. And in addition you will find, if you take the trouble to examine this patient, that there is some wasting of the muscles of the thigh all the way up. The amount of wasting differs in various cases, but in any case, no matter what its cause may be,—and this remark applies to any form of knee-joint disease other than displaced semilunar cartilage, as well as to that condition—when the thigh muscles are markedly wasted, especially if that wasting is excessive, there is invariably “wobbling” of the joint on the affected side. That is a very important fact, since it is impossible to bring about the complete cure of the laxity of the joint without restoring the natural development and tone of the muscles above. There is another point worthy of mention in connection with constant recurrence of irritation in joints from any cause. Constant recurrence of irritation in joints, with or without effusion, always tends to produce an enlargement of the normal synovial folds, and hypertrophied synovial fringes or folds are always liable to be caught between the bones, entering into the formation of the articulation. Recurrent attacks of the kind we are considering not only tend to produce looseness of the joint, but may lead in some respects to actual joint disease—tuberculous disease, for an example, may thus originate. The

proper treatment of these cases is therefore a matter of some moment. This leads me to say that in all operations for the treatment of internal derangement of the knee, supposing the joint to have been opened and a loose semilunar cartilage found, it is not sufficient merely to remove the defective cartilage. The joint should be carefully examined, and any synovial fringes or folds which are of abnormal size or hardness should be taken away, especially from behind the ligamentum patellæ. Removal of the semilunar cartilage will not, in fact, be in all cases sufficient to effect a cure. A considerable number of the cases of recurrence of symptoms after operations for removal of semilunar cartilages are due not to anything which happens to the cartilage itself, but to the fact that the abnormal synovial fringes, which should have been removed at the time of the operation but have been left untouched, are large enough to get between the bones, and so reproduce the exact condition of things which obtained before the treatment. When, therefore, I operate upon this man, as I shall have to do in order to remove the affected semilunar cartilage, I shall take care to see whether the synovial folds behind the ligamentum patellæ are hypertrophied and nodulated to such an extent as to be in danger of getting between the bones. If abnormally enlarged I shall clear them out, leaving a joint free from such possibilities as may arise if the abnormal tissues are left untouched.

There is a great difference of opinion amongst surgeons generally as to whether in these operations a drainage-tube should be used or not. This is rather an important point. Personally, I always use a drainage-tube for twenty-four, or it may be sometimes for forty-eight, hours, according to the condition of the case. I do this not because I am afraid of sealing up the joint, but because the intense pain which many patients suffer in these cases after operation can be entirely obviated by the use of a drainage-tube. If a tube is used so that the synovial fluid, which is generally excessively secreted during the twenty-four or forty-eight hours after the operation, can drain away, practically all the pain to which the patient is liable—provided, of course, that sepsis does not follow—is obviated, an enormous advantage.

So far as the operation itself is concerned I think the risk is very small, but, in spite of what

you read and hear said to the contrary, there occur from time to time a certain number of veritable disasters after this operation; that is to say, the knee may be stiff, it may be completely ankylosed, and, indeed, limbs have been lost afterwards. These cases, however, are so rare in cases conducted by competent persons that they need not be taken very seriously into consideration when speaking of operation. Nevertheless, in discussing the matter with the patient, I think it is your duty in this, as in all other operations, to state that there is a certain risk; it is not fair to put the matter in such a way as to lead people to suppose that there is absolutely no risk in this operation, because, as a matter of fact, there are accidental risks connected with all operations, and it is undoubtedly the right thing to take the patient into your confidence in this respect out of *common fairness to him as well as for your own protection*.

A CASE OF PYLORIC OBSTRUCTION.

This man is suffering from pyloric obstruction, due, I believe, to carcinoma. He tells us that, so far as he knows, he was well until about three months ago. But if you question him a little he will tell you that he had been losing flesh for some time before that date, although he had noticed nothing peculiar about himself until three months ago, when he began to feel discomfort about the stomach, and had occasional attacks of vomiting, especially if he were doing anything which strained him to any extent. This vomiting was very copious, and it has gone on ever since at definite intervals. The notes say that he has constant attacks of vomiting and inability to retain his food for any long period, the amount vomited being very large. He has also had that remarkable difficulty with food which people suffering in this way experience, *i. e.* they feel difficulty in getting food down at all, no matter how hungry they may feel before they commence eating. A little while after taking food the patient feels blown up and uncomfortable. For two months he has been conscious of having in his belly something which is not natural, and for six weeks he has known of the existence of a lump. Since he first noticed this lump it has been growing rapidly in size. Such is the history of his case. If you look at him you will see how terribly thin he is; every rib and cartilage shows, and the elasticity of his skin is

almost gone, though not so defective as you sometimes see in certain cases of carcinoma in the belly. You can see also in his case, which is not always perceptible in these instances, a well-marked tumour, which at present occupies the left side of the belly just under the ribs, moving up and down gently on respiration. This upward and downward movement on respiration is characteristic, but not particularly helpful in the matter of differential diagnosis. All tumours in the belly connected with moveable viscera, whether solid or hollow, will rise and fall to some extent with respiration; but the fact that they do not rise and fall with respiration, as I pointed out to you in another case the other day, is no evidence that they are not connected with moveable viscera, because a malignant tumour or inflammatory mass will, if it lies near any part of the parietes, tend to become attached to the parietes under certain circumstances, and once it becomes attached to the parietes its mobility with respiration ceases, an important point to bear in mind in the diagnosis of these conditions. Mobility with respiration of a tumour of this sort is insisted upon very strongly as being a pathognomonic sign of its connection with some moveable viscus; and it is often said that if a tumour does not move with the respiration that there is a very great doubt as to whether it is connected with a moveable viscus. But under the circumstances I have indicated, it may be quite fixed and may yet have originated in a moveable viscus. In a case of this kind the situation of the tumour may vary greatly from time to time; the position of the tumour will, in fact, greatly depend upon the distension of the stomach—the fuller the stomach the further down on the right side will the tumour be. That is one of the peculiar characteristics of these pyloric tumours; they shift their position with different degrees of distension of the stomach. When I saw this man yesterday, the tumour was near the right anterior superior iliac spine, several inches from its present situation. This tumour is much larger than the average of those connected with disease of the pylorus. As a rule, you must understand, in cases of disease of the pylorus the tumours are small when the patient comes under observation, and are sometimes undiscoverable except under an anæsthetic, and even then are sometimes quite

imperceptible to the touch. Persistent dilatation of the stomach should always direct your attention to the possibility of a pyloric tumour. In making an abdominal section in the case of a patient who has a dilated stomach in which no tumour of any kind can be felt, it is not uncommon when you have the pylorus exposed so that it can be manipulated freely to find a hard, brawny mass, which feels very much like carcinoma. But the condition is not always carcinoma. The question, however, of the differential diagnosis between carcinomatous tumours of the pylorus and fibrous ones is a very intricate one, and rather too large a subject for us to enter upon this afternoon. The diagnosis in the present case, as far as we have gone, is pretty sure. This man has a very large stomach, and he has a very free splash indeed on succussion, which, with the co-existence of a mobile tumour such as you see here, is sufficient to establish the diagnosis. Still it is, I suppose, just possible, you must bear in mind, although all the symptoms which we have mentioned are so very precise, that a tumour of this sort may have been originated in the colon and have affected the stomach only secondarily. But in this particular instance the symptoms are so very obvious that I do not suppose that can be the case. We will assume that our diagnosis is right, and that there is a large mass of carcinoma connected with the pylorus. Some attempt at relieving the symptoms is imperative. The man is fifty-eight years of age, and has become very thin, but he is not altogether in a bad condition for operation. The first point to determine is whether you will operate or not in a case of this sort. In pyloric carcinoma, as in carcinoma of other parts of the intestinal tract, including the rectum, it is not always advisable to operate. It must be remembered that in a case in which the disease has advanced so far as it has here, that an operation is at the best only palliative, and if the patient is in a very bad condition may be associated with so much risk that it is not worth the doing; moreover, in such circumstances so much comfort can be obtained from methodical antiseptic washing out of the stomach that it is preferable to operation in advanced hopeless cases. In this man's case, as he is in fair condition, operation is clearly indicated. Then comes the question of the kind of operation which should be performed.

In this case, seeing the size of the tumour, it is clear that no removal of that tumour is likely to be wise, not because the tumour itself, perhaps, cannot be removed—I have no doubt it can be removed—but because it would be almost certainly impossible to remove the lymphatic infiltration and the secondarily affected glands and so on. The parts about the posterior part of the belly are sure to be involved in secondary disease. If all outlying infiltration, etc., could be removed the obvious indication would be to remove the tumour and as much of the stomach and duodenum as is necessary, together with the secondary disease—in point of fact, to perform a true radical cure, as is attempted in certain cases of carcinoma of the breast, for example. But, following the analogy of the treatment of carcinoma in other parts of the body, we do not, except for very strong reasons, do partial operations for carcinoma. We say that unless we can remove the glands and remove the channels which have borne the secondary disease, operation on the original disease itself under ordinary circumstances is not justifiable.

Therefore, here we have to be guided by the same rule, which practically confines us to an anastomosis between the stomach and small intestine, and this operation (gastro-enterostomy) is, in my experience, almost invariably the only rational treatment in cases of this sort. I have, perhaps, been unlucky in my cases, but the number which I have seen in which pylorotomy could have been considered a justifiable operation has been so small as to be hardly worth consideration. I have seen many cases in which the primary growth could have been removed, but, at the same time, it would have been practically impossible to completely clear away all secondarily involved tissues. We shall therefore operate in this case solely with a view to gastro-enterostomy, opening the abdomen near the middle line; I say *near* the middle line advisedly, because, I suppose, although we still speak of median abdominal section, very few surgeons now operate through the actual middle line, as it is now the custom to avoid making incisions through purely fibrous tissues, as the scars resulting are inclined to be weak, in consequence of the comparatively non-vascular nature of the parts divided, scars resulting from incisions through vascular parts like muscle being much more stable. In former days

every surgeon took care to cut through the fibrous tissue of the middle abdominal line, because it was felt that there was less bleeding, and that there was also less tendency to suppuration there. But in these days we do not care about the bleeding, and we do not fear suppuration. This matter is of less importance above the umbilicus than below, as the tendency to scar-stretching is less above the navel than below it. The abdomen should never be opened exactly in the middle line below the navel.

Having exposed the stomach the practice of surgeons differs somewhat in the choice of the situation of the anastomosis, *i.e.* whether it shall be on the anterior or on the posterior surface of the stomach. Personally, I almost always do an anterior gastro-enterostomy since, in my experience, no special advantage has arisen from the posterior operation, and the anterior is more simple in detail and more quickly accomplished, a very important point in greatly exhausted patients. The posterior operation has one great disadvantage in my experience, inasmuch as the tendency to regurgitation from the intestine into the stomach is certainly much greater than it is in the anterior anastomosis, and I have never had reason for believing that the passage from the stomach towards the intestine is less free in the anterior than it is in the posterior. In some cases, in which adhesions are very strong and extensive, the posterior operation is practically impossible.

This question of regurgitation is one of considerable importance in the early days after the operation. Regurgitation from the intestine into the stomach, if occurring to any material extent, is invariably followed by passive regurgitation from the stomach through the gullet and mouth (sometimes improperly called "vomiting"), a most distressing condition of affairs, which tends to the production of septic broncho-pneumonia, and prevents any feeding by the mouth. This immediate regurgitation can be generally entirely avoided by placing the patient in the sitting position in bed, instead of flat, as is too often done. I was, I believe, the first to adopt this method, which has now become almost universal. With regard to the means used for effecting the anastomosis there is naturally much variance of opinion amongst surgeons. I almost invariably use Senn's plates, because I have never been disappointed with the

immediate results I have obtained, and have had no evidence of any harmful contraction of the opening subsequently. My experience of Murphy's button is not good enough to lead me to continue its use further, and simple stitching is unnecessarily long in completion.

With regard to prognosis, patients who have had gastro-enterostomy done for *carcinoma*, of course do not live long. If this man lives three months it is as much as we can expect. But there is a hopeful point about some of these cases, in that the disease sometimes turns out not to be carcinoma; it is, in fact, sometimes absolutely impossible to say whether the tumour is carcinoma until you have seen a section under the microscope. Some of the cases which seem to offer the worst prospects before operation turn out in the end the best. I operated upon a man nine years ago who is still alive, the case being the longest one on record, as far as I know, after gastro-enterostomy for what appeared to be an undoubted carcinoma of the pylorus. The tumour shrivelled after the operation, so it could hardly have been carcinoma. The patient was a man named C—, and his case afforded the first operation of gastro-enterostomy which was performed in St. George's Hospital. One point in the diagnosis of pyloric obstruction I have forgotten to mention. It is said that the presence or absence of free hydrochloric acid in the stomach is a very important symptom in the diagnosis of doubtful cases of pyloric obstruction—I am not speaking only of carcinomatous obstruction, but any obstruction of the pylorus, whether from carcinoma or from fibrous constriction. It is held by some people that if in a doubtful case of dilatation of the stomach there is an absence of free hydrochloric acid in the viscus, there is absolutely certain to be gross obstruction of the pylorus. With this view I do not quite agree, as it is not in accord with my experience; as a piece of additional evidence with other symptoms the absence of free hydrochloric acid is undoubtedly corroborative, but that, standing alone, it is a certain evidence of obstruction I very much doubt. As you know, however, I am not much given to rely upon any of the so-called pathognomonic symptoms by themselves.

CYSTIC BRONCHOCELE.

Here is a girl, 16 years old, of the anæmic

type. You can see at once that there is a well-marked increase in the size of the lower part of her throat, the enlargement being more prominent on the left side than on the other. In point of fact, the tumour is evidently connected with the thyroid gland; it rises in swallowing with the gland, and is clearly a part of it. It is therefore a form of goitre, and the first indication is to decide as to its nature if possible. The whole of the gland is obviously enlarged, and you may therefore think at first sight that we have to deal with one of those conditions of the thyroid gland which are called parenchymatous enlargements, that is to say, uniform general enlargement, almost always associated with the anæmic type of patient, and occurring very often at the time of life of this girl. In this case, as is not uncommon, the disease is hereditary, the girl's mother having had the same condition of things. The hereditary element in the case, the aspect of the tumour and its shape, would lead you to think it was a parenchymatous goitre, without exophthalmos, *i. e.* without protrusion of the eyeballs, which is often seen in these cases at this time of life. Upon manipulating this goitre you will find that the appearance of uniformity in enlargement is deceptive, and that the major part of the large side of the goitre is made up of several nodular masses, conveying to the touch a sensation like that of tense cysts. As a matter of fact, very little of the goitre is parenchymatous, the bulk of the tumour being composed of cysts—the case is one of cystic bronchocele. The patient suffers no disability in swallowing or breathing at present, but the tumour is rapidly increasing in size, especially on the larger side, and the oncoming of dyspnoea, etc., is only a matter of time. Already there is a little hoarseness in the voice which is recent, and is a sure sign of further troubles to come. The change in the character of the voice in these cases is a most important indication of the coming of other troubles. It is clear, therefore, that some energetic treatment must be adopted, as such medicinal and palliative measures as are available have been tried, and, as might have been expected, have been found to be useless. The only remaining treatment is operation, and three operations are available—(1) enucleation of the cysts; (2) enucleation resection, and (3) resection of the left half of the thyroid body. Of these I have no hesitation in selecting

enucleation; it is by far the least severe, and although the enlarged portion of the gland itself will not be removed at the operation, the removal of the cysts will be followed surely by complete shrinkage of the abnormal thyroid tissue. I am quite certain that the less the thyroid is removed in these cases, especially in young people, the better, for although the removal of half the thyroid is generally held to be harmless so far as any constitutional effect is concerned, I am convinced that remote effects, which are commonly overlooked, not infrequently follow. In enucleating these tumours, whether they are cystic or solid, let me warn you not to be misled as to the ease of the operation by the way in which the books speak of "shelling" these tumours out, as it is very rarely that they "shell out" in the ordinary sense of the word, and you must be prepared sometimes for very considerable difficulty in the enucleation, and you may find in some cases that a considerable amount of blood is lost. I mention these points because I find that candidates in examinations in speaking of "shelling out" these tumours seem to believe that the process is akin to the shelling out of a freely moveable sebaceous cyst—a belief which, if uncorrected, may lead a man into grievous trouble in his first operation of this kind. The whole secret in enucleating with as little difficulty as possible is, of course, to avoid making any attempt at getting the tumour out before it is actually exposed by the free division of its true capsule, a thing which, to the inexperienced, is not always easy to be sure about.

Quinine Rashes.—In the diagnosis two points are of special interest—the involvement of mucous membranes and desquamation. These facts bring scarlet fever into the question of diagnosis. Treatment is symptomatic. When the idiosyncrasy is known, the drug should be given hypodermically. The differential diagnosis between scarlet fever and quinine rash should be based on the history of exposure to scarlet fever, the greater intensity of constitutional disturbance in scarlet fever; the desquamation in quinine rash is not so persistent, there is not that peculiar desquamation of the fingers, especially around the nails, in quinine rash, there are not the enlarged tonsils covered by a yellowish exudate, as in scarlet fever, there is not apt to be the strawberry tongue in quinine rashes. *Medical News*, November 23rd, 1901.—*Monthly Cyclopædia*, January, 1902.

A CLINICAL LECTURE ON CRETINISM AND ALLIED CONDITIONS.

Delivered at the Hospital for Sick Children, Great
Ormond Street, W.C., December 9th, 1901.

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LADIES AND GENTLEMEN,—With this lecture, the first of the series that are given at this hospital during the winter session will be concluded, and as we are so close upon the festive season of Christmas I have ventured to choose a subject which is more cheerful than most of those with which the physician has usually to deal. Among the more recent advances of medical treatment none has been more brilliantly successful than the treatment of cretinism, and when we sit, as did that famous prophet of yore, in despair upon a hill-top with our heads upon our knees, we are, I think, roused to fresh efforts by the sight of a small piece of a thyroid tablet.

Let me then first do honour to some of the distinguished names that are associated with the discovery of this treatment—to Mr. Victor Horsley, Dr. Murray, Dr. Hector Mackenzie, Dr. Hale-White, and Dr. Howitz. These are some of the names of the most distinguished workers on the subject of thyroid feeding in myxœdema and cretinism. I need hardly remind you that cretinism is dependent upon a disorder of the thyroid gland, and also that there are other diseases and maladies which are associated with disease of the thyroid gland. Thus, with the over-action of this gland we associate exophthalmic goitre. With diminution of function we associate myxœdema in the adult and cretinism in the child. It is interesting that although myxœdema and cretinism are, in their pathology, almost identical, you find that clinically there are some points of distinction between them. Such points of distinction as there are will be easily explained by the difference of the age incidence in the two affections. In myxœdema the chief feature is the loss of function, while the chief feature about cretinism, I suppose, is non-development of function. For instance, a woman of fifty or sixty years of age who develops myx-

œdema, and who was previously bright and active in mind, with the onset of the disease becomes mentally dull. On the other hand, the cretinous child is dull and heavy from the first, and if that child be untreated the mental condition never develops, neither does the physical condition. That is the first point of distinction between myxœdema and cretinism. Another is that the sexual functions do not develop in the cretin. These both depend on diminution of function of the thyroid gland. Another series of conditions associated with the disorder of the thyroid gland are goitres—goitres without definite symptoms—simple goitres as they are called. Yet another are tumours of the thyroid, about which I have nothing to say to-day.

CRETINISM.

As you can understand, since the condition of cretinism is not very common, and now that it is well recognised and treated, it is very difficult at the right moment to get a case which shows the characteristics. Unfortunately the only case under my care which showed these characteristics, and which had not been treated for any length of time, is too ill with bronchitis to come to-day. I must therefore ask you to be content with this picture of the characteristic cretin, which I will hand round.

The method of demonstration I shall adopt will be this: I will first point out to you some of the most important symptoms of cretinism, then deal with the prognosis, and then with the points in the treatment—for there are very important points in regard to treatment—and lastly, I will show you one or two cases which very well illustrate the results of treatment.

Of cretinism, two forms are described. First of all endemic cretinism. This is a condition which is met with in the valleys of mountainous districts, and it is usually associated with goitre. On the other hand, there is the sporadic cretinism which we see in England, which, as a rule, is not associated with goitre, but with a diminution, or complete absence, of the thyroid gland. Then there is one other form, namely, juvenile myxœdema. Juvenile myxœdema is a condition of this sort. A child of seven or eight years of age, who was in perfect health, suffers, perhaps, from a severe infectious disease, and as a result of this there may

be some inflammation or injury to the thyroid gland which destroys the function, and from that time myxœdema develops. In other words, the course of the disease resembles myxœdema in adult life. I do not think there is much need to distinguish between juvenile myxœdema and cretinism.

Now let us consider the characteristic cretin. The cretin is dwarfed in stature, and the older the subject the more this strikes you. In the case pictured here, in Dr. Murray's book, the woman was twenty-eight years of age, yet she was only thirty-four inches tall. I show you another picture of her after she had been treated. Another point is that cretins are not only short in stature, but their skin is harsh and dry, and they are backward mentally as well as physically—so backward that in bad cases they can only just walk, and very often can only hang on to a chair. The face is very characteristic: it is usually pale, not red, the forehead is wrinkled, the eyes wide apart, the nose depressed. The tongue is large, and is often protruded from the mouth. The head is large in the posterior part, and the hair is scanty and harsh. When the patient is grown up the voice is guttural and monotonous. There is great stunting of the limbs, and the hands are thick and podgy. In many of the cases there is an umbilical hernia, and the abdomen is usually protuberant. If you take the temperature you will generally find it is sub-normal.

Next let us notice the mental condition. These cases are always very backward, but in some instances this defect is more pronounced than in others. In some cases you find that the child scarcely recognises anyone. More usually the child will recognise its mother, and sometimes common objects. As they grow up there is no sexual development; they do not grow to any extent, nor does the mind develop.

I have so far omitted one point, because I want to insist upon it, namely, the very characteristic œdema, which is more marked in the lax tissues, such as under the eyelids, and especially above the clavicles. These masses of solid œdema above the collar-bones on either side give rise to conditions which are known as fatty tumours. Dentition also is late in these children.

As a rule, the condition is not recognised at birth; generally the child is some months old

before the state is noticed ; indeed, it may be that two or three years have elapsed before the mother has her attention called to the fact that the child is backward. That is an important point to bear in mind.

When untreated the prognosis is hopeless as to any real mental or physical improvement. Not only that, but these subjects usually die young. Untreated cretins seldom grow up ; it is very unusual to meet with a case like this of Dr. Murray's, who was *æt.* 28. These cretinous children generally die of broncho-pneumonia or tuberculosis, or of some infectious disease. So the prognosis in the old days was very bad. But since the introduction of the thyroid treatment the prognosis is different, and I should like to insist upon a few points in the prognosis as it stands now with the thyroid treatment.

The first point to ascertain is the length of time the symptoms have been going on before you see the patient. You can easily understand that a child which is cretinous, and which has been allowed to remain in a cretinous state for several years, will not improve as much as you would wish under the thyroid treatment. You must remember that during all those years the brain has not been receptive. Secondly, you have to remember that in some cases the physical condition of the cretin is more backward than is the mental, and in others *vice versa*. I have here a child whose mind is fairly bright, but it is very definitely cretinous as far as the growth and the condition of skin and hair are concerned. I have had a case illustrating the converse, in which the mind was almost a blank, and yet the physical condition of cretinism was not very marked ; but in the majority of cases the physical and mental conditions are approximately altered in due proportion. If the mental condition is only slightly altered, and you get the case early, the prognosis is very good ; these children do wonderfully well, and some of them get on at school. On the other hand, a considerable number of them improve physically under treatment, but mentally remain backward, as in the case of a little girl whom I have under care now. They are, with the thyroid treatment, able to resist infectious diseases, and I have two cases here which have been attacked with diphtheria and have survived, and both will, I hope, grow up as strong children. In the matter of prognosis, then,

a great deal depends upon how soon you begin the treatment and upon what is the mental condition. Remember that the mental condition always improves more slowly than does the physical.

Now, with regard to the treatment itself, it practically consists of feeding by means of thyroid preparations. I will first remind you of the pharmacopœal preparations. There is the *Liquor Thyroidii*, \mathfrak{m} 100 of which is equivalent to one healthy sheep's thyroid. The dose of that is \mathfrak{m} 3 to 15. Then there is the dry preparation of thyroid, the strength of which is not accurately known, and that is given in doses of 3 to 10 grains. Then there is what is looked upon by some as the essential principle, iodo-thyrine, that is to say, iodine in some organic combination, made up with sugar and milk, the dose of which is about 30 grains. The essential principle, whatever it is, seems a fairly stable body, and most of the methods of administering the drug will meet with success. In this hospital we often use the tablet form.

It is a powerful remedy, and therefore wants using with care, and I feel that I cannot do better than follow Dr. Murray in describing the treatment in stages. The first thing is to find the maximum dose which the child can bear without developing symptoms of thyroidism. Always begin with a small dose, because we do not know exactly the strength of the preparation, neither do we know at first how much the particular child will stand. Then it should be run up to the limit—I will mention in a moment some indications that the limit has been reached. You should then continue the maximum dose until you have made up your lee way, that is, until you have corrected the symptoms. Then the third stage, which, so far as we know, lasts throughout the rest of life, consists in diminishing that maximum dose down to the limit which is consistent with an absence of return of the symptoms.

The next thing is to know what are the symptoms which will make you suspect you have gone a little too far ; you do not meet with them, Dr. Murray points out, so frequently in cretinism as you do in the myxœdema of adults. Some of the important symptoms of an overdose are these :—Mental excitement and irritability, rise of temperature, obscure pain and palpitations, with, some-

times, urgent dyspnoea. Those remind you that you have got to an overdose, and, as Dr. Cheadle pointed out to me, you are generally at about the right dosage when the drug causes a slight rise of temperature. If you get beyond that dosage you get the symptoms of thyroidism, whereas if you get any distance below that dose you will not get the full result.

There is one danger in the treatment which in the child is fortunately absent, but which you will have to bear in mind with regard to myxoedema. If you treat with thyroid gland an adult suffering from myxoedema she improves very rapidly, and the result is she becomes energetic instead of dull, and will try to do too much. Now Dr. Murray has pointed out that the heart suffers in myxoedema, and that there is atrophy of the heart muscle. The result is that over-exertion by such a patient when under treatment may result in a sudden attack of syncope. That danger, as I have said, we do not meet with in children. In the cretinous child the heart is a young one, and can bear the strain. As to this atrophy of the heart muscle in myxoedema, I have made one post-mortem on such a case, and found by microscopic investigation that the atrophy was exceedingly marked. But there are other little difficulties to look out for, and one of these is bending of the legs. When these children begin to get about sometimes the legs begin to bend, and then you must see that the child is taken off his feet and rested until the legs are stronger. Another point you must be careful about is to keep them warm, because they very readily take cold. Those are the chief points in the treatment of a cretinous child. I will now show you the cases.

This little boy first came to see me in April, 1900. Mentally he was bright for a cretin, but the physical symptoms of cretinism were very marked. The skin was very harsh and rough, and there was some eczema, for which the child had been treated at a London hospital, but without any improvement. In April, 1900, I commenced to treat him, beginning with a grain of the thyroid tablet, at first once a day, then twice, and later three times a day. Improvement was rapid, but in the course of 1900 the mother left off the treatment. The child was brought back to me almost a year after my first acquaintance with him, that is, in April of this year, and then all the eczema had

come back, and the child was really a well-marked cretin. Now you would scarcely have recognised him as a cretin at all. The hands are still a little thick, but that is all. He has grown, and, the mother says, is much sharper and brighter. About the first thing which you will notice as a result of this treatment is a subsidence in the swelling of the tongue, and then that the skin will become smooth and soft. This child's skin is now quite smooth, and the hair has grown well, the eczema having disappeared. He was three years of age when he came, but he has only been able to walk during the last six months. The prognosis in such a case as this is good, and it illustrates how quickly an eczema will disappear under this treatment when there is a cretinous condition behind it.

Probably you would not recognise this next little girl as a cretin. She is *æt.* 8½ years, and has been under treatment four and a half years. Although very shy and rather backward, she has recently earned a certificate for excellence of work at school, which her mother brings to show us. She never was a very marked cretin. The interesting point about the case is—and this introduces the most interesting part of this demonstration—that the child had an attack of diphtheria some months back, and while undergoing treatment for the diphtheria the thyroid was discontinued for five weeks. What happened then was that a swelling appeared in the neck, not very large, but quite definite, in the position of the thyroid gland, and the child became decidedly more dull and slow in her speech. Those were very suggestive symptoms. When I saw her the mother had recommenced the tablets, and now the swelling has disappeared. So here is a child who was only slightly cretinous at one time, and is now improved so much that no one would know her as a cretin. But she is still rather backward, and probably will always be a little dull. This introduces me to another case, which, unfortunately, I cannot show you to-day.

The patient was a girl *æt.* 8, who had been under treatment five years, and when I first saw her I should never have recognised that she was a cretin if I had not known her history. The condition had been originally one of partial cretinism, and it seemed possible to me that while the child had been growing up the thyroid might have deve-

loped also. So I ventured to cautiously diminish thyroid treatment, until I gave it up altogether. Two months afterwards the mother brought the child to me with a large goitre. The same phenomenon, you see, was noticed, but to a more pronounced degree, as in the case we have just seen. I showed her to several doctors at the clinique that morning, and they all saw this large goitre. With the swelling of the thyroid there had developed slowness of speech and dulness. Thyroid treatment was at once resumed, and then the enlargement of the gland disappeared. That is a very interesting and important observation. And now I think I may allude to some of the experimental work which has been done upon the thyroid gland.

With sporadic cretinism, as already stated, we usually associate atrophy of the thyroid, but clearly in these two cases, although the thyroid was not functional, it was not completely atrophied. It is very suggestive that this sudden enlargement of the thyroid, when the thyroid treatment was stopped, was compensatory, but though it was compensatory it was not effectual. The child developed symptoms of cretinism, although the thyroid did enlarge, and although this enlargement in the second case reached a considerable size. Tempting though it is to say this enlargement is compensatory, it is in some degree an assumption, for the function of the thyroid is not quite so simple or easy to understand as we might imagine. We know that when goitres were removed experimentally—Kocher, of Berne, pointed this out—that this total extirpation of the thyroid in man was followed by an acute myxœdema which proved fatal. In monkeys and dogs total ablation of the thyroid is followed by fatal myxœdema. But in the case of the rabbit, if the thyroid is removed, in a considerable number of cases there is no change, from which you might infer that in this respect the metabolism of the rabbit was different from the metabolism in man. But that is not the explanation. The explanation lies in the fact that in addition to the thyroid you have to take into account the parathyroid. In rabbits the parathyroid is at some distance from the thyroid, whereas in man the parathyroids are so closely connected with the thyroid that in the removal of the latter you take away the parathyroids also, and there is thus produced a fatal myxœdema. In

rabbits the parathyroids are left, and fatal myxœdema does not result. If the thyroid only is removed in the dog and monkey and the parathyroids left—a very difficult operation—these animals do not die in the rapid way they do when the entire thyroid and parathyroids are ablated. If only one parathyroid is left in the dog the animal will live. So the parathyroid must have some very important function in regard to the secretion of the thyroid gland, and that is why it is rather an assumption to say that the increase in the size of the thyroid in these two cases was a compensatory phenomenon. The practical importance of that observation is that it seems to throw light upon certain cases of simple goitre which get well when treated with thyroid gland. Anyone who is interested in this subject I might refer to the Erasmus Wilson Lectures by Mr. Edmunds in the 'Lancet' of this year (1901), for these tell you a great deal about the experimental aspect of the subject. In looking over these lectures I found it was stated that the best treatment for some of those cases of goitre without symptoms is treatment by thyroid gland, and with this treatment recovery may be complete. Another interesting point about these simple goitres is that they may relapse on cessation of this treatment. Now, I may say that if I had been shown the second of these two children, and I think if you had seen her without knowing anything about the history, and with this goitre, we should not have recognised her as a case of cretinism at all. And if I had treated her with thyroid gland in ignorance of the history of cretinism it would have been pronounced a case of goitre cured by thyroid treatment. I think, then, it is possible that these goitres without symptoms which are treated by thyroid and get well may possibly be associated with slight cretinism, which may not be detected, or even be capable of detection, except by the result of that treatment. These cases seem, then, to provide a very interesting link. To recapitulate, we have here a child who was under Dr. Garrod before me, who was known to be slightly cretinous, and had not an enlarged thyroid, and who was, on theoretical grounds, thought to have a diminished thyroid or an absence of that gland altogether. This child was under treatment five years. The treatment was left off for two months, and then a large goitre formed. The resumption

of the treatment by thyroid extract completely cured this goitre.

THE MONGOLIAN IMBECILE.

The title of my demonstration was "Cretinism and Allied Conditions;" and in choosing this I was more influenced by practical than by scientific considerations. There is no doubt that practically there are other conditions which we sometimes mistake for cretinism because they bear a superficial resemblance, and that if we make a prognosis in these cases which is based upon that for cretinism, we get into difficulties. One of the conditions I have in mind is the Mongolian type of imbecile. I shall adopt the same procedure in regard to this condition as I did in speaking of cretinism. I will describe the characters of the affection, and in giving this description wish to express my indebtedness to a very interesting pamphlet by my colleague, Dr. Still, and then I will show you some cases. The Mongolian idiot or imbecile is short in stature, backward in dentition, and backward mentally, as is the cretin. The face also shows some of the characteristics of the cretin, but yet it differs. Thus, the palpebral fissures slant, and the cheeks are usually red, while those of the cretins are pale. Again, the head of the cretin is large behind; the head of the Mongol has very little back to it, and is broad across the vertex. The palate is often highly arched and narrow; the tongue, as is that of the cretin, is generally large, though not invariably so, and it is not, as a rule, so large as in the cretin, and in older children of this type has a most curious fissured and hacked appearance. Then, again, the hair of the Mongol differs from that of the cretin, for, as a rule, it is natural, whereas in the cretin it is scanty and harsh. There are no fat pads, nor is there oedema. The hands, however, are curiously broad and thick, and the little finger sometimes curves towards the ring finger. The limbs are short. The mental state is interesting. These children are very backward, but as a rule they are more lively than the cretinous child. They are also fond of music, and after they have learnt to walk they like to dance whenever they can. The voice is guttural, like that of the cretin. The nose of the Mongolian is very much depressed, as is also that of the cretin.

The prognosis in Mongolian imbecility is, I am

sorry to say, not good. If you take as the test of usefulness the ability to earn a living, then these children are not successful, and usually they cannot be trusted to do anything. Still, they are often affectionate, and they know their relatives, but not very much more. Neither do they react to treatment by means of thyroid to any extent; but I include myself amongst those who believe that thyroid may do a little good in these cases, but you must be careful not to promise a good result from it. These children are delicate, and they seldom live long. The extremities are generally cold and blue, and they fall easy victims to tuberculosis or broncho-pneumonia, and, as Dr. Garrod has shown, a considerable number have congenital heart disease. This week a Mongolian imbecile under my care died who had congenital heart disease. These children should be carefully kept warm; a trial by thyroid may very well be given, and, if possible, they should be carefully educated at special schools. I will now show you some examples. This boy is *æt.* 10 years. You see he has got great thick fingers, and that they are blue and cold. The slant of the palpebral fissures is well illustrated. You will notice his head is small, and there is very little back to it. His tongue is not particularly big. His voice is low pitched and monotonous; I am sorry to say he has got severe spinal caries from tuberculosis, and this great curvature may help to impress upon your memory the delicacy of these children.

Here is another child whom you might possibly mistake for a cretin, but, you see, the face is red, the hair is silky, not harsh. The tongue is big, and is kept out of the mouth. The child snuffles a good deal. There are the slanting palpebral fissures and depressed nose. The older children have a guttural voice, but the young ones sometimes crow, and you might mistake their noise for laryngismus stridulus. Notice here the curving of the little finger towards the ring finger. There are no fat pads, and the skin is not harsh and dry. This child seemed to improve a little on the thyroid treatment, but not to any great extent.

Here is another child, with very large ears; he is a very bright little chap, he is very fond of music, and he dances, and is a great mimic of older children. You will notice that the slant of the palpebral fissures is very marked in this case; the skin is smooth, but the head is characteristic of

that of a Mongol. He also shows the outward curve of the little finger.

ACHONDROPLASIA.

The next condition I have to speak of is rare, and I could not get an example of it, but can show you very good photographs of the condition, kindly lent me by my colleague, Mr. T. H. Kellock. We do not know very much about achondroplasia; in fact, we know less about the pathology of the disease than we do of Mongolian imbecility, so, after the fashion of our profession, we give it long names. It is a condition of osteo-genesis imperfecta, that is to say, a condition in which there is imperfect bone formation. It is also sometimes called osteo-psathyrosis, and Chondrodystrophia foetalis. Another name is periosteal aplasia, and a fourth is foetal rickets, and yet another is this I have chosen, achondroplasia. Most of the children who are the subjects of achondroplasia are born dead, but some of them survive, and it is these you are apt to mistake for cretins. They are short in stature and very curious in appearance, but they are intelligent, and that is the great difference between them and cretins. They have broad heads, long bodies, and short limbs. You sometimes see them about the streets; they are those waddling, intelligent dwarfs who are sometimes found sweeping crossings and engaged in other useful employments. There is apparently an arrest of ossification of the bones or some imperfection of bone formation, and a great feature is the shortness of the long bones. One other point which you see in the photograph is that the fingers are divergent, and cannot be quite approximated. These children do not grow, and you can do nothing with that deficiency by way of treatment. So it does not do to say in these cases that if you treat them with thyroid they will grow six inches in a year.

INFANTILISM.

One other condition sometimes mistaken for cretinism is infantilism, which in some cases is due to congenital syphilis. These children are backward in every way, but they are not cretins or Mongolian imbeciles, and do not improve under thyroid feeding.

ANOMALOUS CASES.

We are still left with a certain number of cases in which you suspect there is something of the cretin, a something which is difficult to put into words; backward children in which you think there must be some cretinous element. What are you to do? The rule to adopt is to treat them with thyroid, only remember that in doing so you are using a powerful drug, and if there is nothing cretinous about the case it will quickly show symptoms of thyroidism. Cretins who are treated with thyroid generally improve rapidly, so I should say that improvement will be noticed in three to five weeks after commencing treatment, and be first manifested by the tongue becoming distinctly smaller. Therefore if you get no results from the thyroid, except that the child becomes thin and miserable, it is certain that there is nothing cretinous about it. If I have been able to-day to assist you a little in the management of this curious group of cases, I shall feel that our time has not been wasted.

Perforation in Typhoid.—Dr. H. M. Taylor says: Greig Smith, J. B. Murphy, and Osler have all laid stress on the idea that an abolished peristalsis and cessation of gaseous movement point to perforation. Murphy and Greig Smith notably regard this evidence as of great diagnostic value. My experience prompts me to attach most importance to (a) pain, (b) muscular rigidity, (c) inhibited peristalsis. As we have outlined, we need greater proficiency in diagnosis far more than improved technique. When we operate and how to operate need not detain us. If there is shock from perforation, a majority of operators will wait for reaction; others contend that every hour's delay means more extravasation of intestinal contents. If there is no shock, or when the shock is not dangerously profound, immediate operative intervention is indicated. Dr. Keen's statistics show that a larger number of patients recovered when the operation was done in about twelve hours after perforation—presumably after reaction from primary shock, but before the advent of shock of sepsis. The operative technique does not differ from that indicated in other acute peritoneal infections. We should bear in mind that the perforation is usually single, and is more frequently within the last eighteen inches of the small bowel.—*New York Medical Journal*, February 1st, 1902.

THE VARIETIES OF DILATED STOMACHS.

A Post-Graduate Lecture delivered at the West London Hospital.

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In a former lecture I fully discussed the difference between distension and dilatation of the stomach, and I also referred to the normal position of the organ; I shall now only briefly refer to these two points.

In the first place, I am strongly of opinion that in its normal position in health the stomach is placed in a nearly vertical direction, and that the pylorus is absolutely the lowest point in the stomach. The two chief arguments in favour of the vertical position of the organ are, firstly, that after swallowing corrosive poisons, the resulting ulceration is found to be confined to the neighbourhood of the pylorus, whereas if the stomach were placed horizontally, the centre of the lower border would be affected; the second argument is that at least seven eighths, and sometimes the whole of the stomach is to the left of the middle line, and that the pyloric and the cardiac orifices are separated by from three to five inches in a vertical plane.

The main differences between distension and dilatation of the stomach consist in the portion of the organ in which the enlargement takes place; after a full meal the stomach is uniformly enlarged, and although the stomach becomes more oblique than vertical, the pylorus remains the lowest part in the stomach; in dilatation, on the other hand, the portion of the stomach known as the pyloric antrum is principally affected, with the result that the lower border of the dilated part reaches a level very much below the pylorus, and so is incapable of becoming emptied. This accounts for dilatation in cases where there is no stenosis of the pylorus, and in which the trouble is caused by stagnation of the stomach contents, which in its turn produces hyperacidity and a chronic gastritis, with further dilatation.

The following are the principal causes of dilatation of the stomach; I have classified them according to their cause, into three chief groups:

a. Causes in relation to gastric ulcer.

b. Causes in relation to new growths.

c. Other causes.

Under the heading *a* we have the following causes:

- (1) Cicatricial contraction of the pylorus itself.
- (2) Adhesions round the pylorus or in its neighbourhood causing a kink, due to old gastric ulcer.
- (3) Spasm of the pylorus, due to presence of an ulcer either near the pylorus or in any distant part.
- (4) Hour-glass contraction of the stomach.

Under heading *b* we have:

- (1) Carcinoma of the pylorus or of the stomach extending into the pylorus.

- (2) Adhesions round a growth in the stomach, causing a kink.

- (3) Obstruction by a polypoid tumour which becomes fixed in the pyloric opening.

- (4) Simple tumour of the pylorus. This is practically confined to children, and is often congenital.

Under the heading *c* we have seven different causes:

- (1) Spasm of the pylorus, due to excess of free hydrochloric acid.

- (2) Adhesions in the neighbourhood of the pylorus, due to any of the following causes: (*a*) inflammation in the gall-bladder, (*b*) abscess of the liver, (*c*) perinephritic abscess on the right side, (*d*) abscess of stomach-wall, caused by foreign body such as a fish-bone, (*e*) gumma of liver and any other inflammation in the neighbourhood.

- (3) Traction on the pylorus by means of a floating kidney on the right side.

- (4) Traction by means of an irreducible omental hernia.

- (5) Gastroptosis.

- (6) Various neuroses.

- (7) Acute dilatation, due to azotic poisons.

By far the most important classes in this list are the first two, but I do not propose to discuss cases of malignant disease to-day.

The most common of the causes of dilatation dependent on gastric ulcer is cicatricial contraction in the region of the pylorus. In such cases the pylorus may become so narrowed as only to admit a fine probe, while its walls are so rigid as to prevent any attempt at dilatation; in fact, the cicatricial tissue in some cases is as hard as a scirrhus tumour. Next in importance is the dilatation due to adhesions in the neighbourhood of an

old ulcer pulling upon the pylorus. These two causes have been recognised for a long time, but the dilatation due to spasm of the pylorus is not so generally admitted. However, it is a fact that the presence of a gastric ulcer, in whatever part of the stomach it may be situated, causes an increase in the amount of free hydrochloric acid, and this produces a spasm of the pylorus. The continued spasm of the pylorus is sufficient to cause the initial stage of dilatation, and when this has occurred, the inability of the stomach to empty itself of its contents causes decomposition of the residue of the stomach contents, with consequent increase of the dilatation, which will continue even though the original spasm has passed off. This is an important fact, since it will explain those cases of so-called idiopathic dilatation of the stomach, where at an operation, considerable dilatation is found without any stenosis of the pylorus. In the same way it is easy to understand that a similar chain of events may occur in cases of hyperchlorhydria which are not associated with ulcer. Owing to a long-continued gastritis, often associated with bacterial poisoning caused by the swallowing of pus from oral sepsis, an excess of free hydrochloric acid occurs, and this produces spasm of the pylorus, with resulting dilatation as just explained.

In the class of cases due to adhesions, the most frequent cause, excluding gastric ulcer, will be found to be inflammation of the gall-bladder or ducts; in fact, it is very seldom that pericholecystitis occurs without producing some adhesion to the pylorus; indeed, I have noted adhesion to the pylorus in every case of cholecystitis. In one case on which I operated, the dilatation of the stomach was due to adhesion to a gumma of the liver, and was cured by a pyloroplasty.

The next cause to which I would draw attention is traction produced by a floating right kidney. At first it may seem rather difficult to see how this acts; it is found, however, that there is a band of fibrous tissue connecting the upper part of the right kidney to the first part of the duodenum; it therefore follows that any considerable displacement of the kidney will cause traction on the duodenum by means of this band, and will produce a kink at the pylorus, with consequent dilatation.

This was well shown in the case of a young girl *æt.* 20, who was sent up to me from the country with symptoms of dilated stomach and fainting

fits. On examination I found that the stomach was dilated, reaching to the level of the umbilicus, and there was a freely movable kidney on the right side.

In the absence of any other cause, I gave my opinion that the floating kidney was the cause of the trouble, and I sent her to the hospital and stitched up her kidney, with the result that the dilatation of her stomach and her other symptoms disappeared.

Another interesting condition is kinking of the pylorus, caused by traction of an irreducible omental hernia, which is readily understood when the piece of omentum is attached near the pylorus. An interesting case illustrating this condition is described by Bennett. A man *æt.* 40 had suffered for some years with dyspepsia, which was relieved by eructations, and latterly by vomiting of large quantities, which occurred about every third day. The stomach was found to be greatly dilated, and a small omental hernia was found at the umbilicus; this was tender, and pressure over it caused nausea. The hernia was explored and found to consist of a small pedunculated piece of omentum which went back with a spring directly the ring was divided. The dilatation of the stomach subsided, and the patient was completely relieved.

The dilatation due to gastropotosis can usually be distinguished from the other causes by the co-existence of prolapse of the other viscera, more especially the liver and kidneys, and so does not call for any special mention. In many cases the dilatation is only apparent and not real, since the lower border of the stomach may be below the umbilicus without any definite dilatation.

Next I would draw your attention to acute dilatation of the stomach, which is usually due to ingestion of some ptomaines. It is a rare and very serious condition, and is usually rapidly fatal. In such cases the stomach becomes suddenly dilated, and usually causes so much pressure on the heart as to produce syncope, this result being also contributed to by the absorption of ptomaines. The pathology of the condition has not been thoroughly worked out.

It must not be considered that dilatation of the stomach is confined to adult life, since a fair number of cases of cancer of the pylorus have been recorded in infancy, and a much larger number of cases of congenital fibrous stricture of the pylorus

have recently been published. In such cases the child emaciates rapidly, and a tumour can often be felt in the pyloric region. Most of the cases have died, and the condition has only been recognised at the post-mortem, but one or two have recovered after Loretta's operation or after gastro-enterostomy.

On Disorders of Assimilation, Digestion, etc.—Sir Lauder Brunton has recently issued a volume entitled, 'On Disorders of Assimilation, Digestion, etc.,' published by Macmillan and Co. The book is a collection of papers and addresses, and in the one dealing with diabetes Sir Lauder Brunton gives an account of what he believes to be the first attempt to show that solid organs other than secreting glands contain a ferment, or, as it is now called, an enzyme, by the action of which tissue metabolism within the body is carried on. The following remarks on alcohol show the practical tenor and excellence of the contents of this interesting volume :

Among well-to-do people gastritis of a superficial catarrhal character is chiefly found, and enteritis occurs, especially among wine merchants. In fact, the man who never gets drunk, but who is continually drinking, has almost always loose and never firm motions. This enteritis and this liquid condition of the motions may last for twenty or thirty years; the motions are catarrhal and not dysenteric. On the other hand, the workman who gets drunk once or twice a week, but hardly touches liquor at other times, suffers rather from constipation. The well-to-do drunkard often suffers from a tendency to bleed from the nose or from the stomach, from the kidneys, or from the liver; he has also a tendency to atheroma. Among rich people glycosuria may occur at the age of thirty-five years. In such persons enteritis is very serious, and is accompanied by hepatic complications, such as hypertrophic cirrhosis, which may last seven or eight years, while such affections are very rare in the artisan.

The effects upon the nervous system of wines, beers, and spirits are due—first, to their reflex action on the circulation; and, secondly, to their effect upon the brain and nerve-centres after absorption.

The stimulant effect of spirits upon the circulation is greatest, and that of beer is least.

The action upon the nerve-centres is also most rapid in the case of spirits, and slowest in the case of beer. On this account, the restraining powers of the nervous system being paralysed, while the lower centres are still undergoing greater stimulation, there is a greater tendency to wild excitement from spirits than from either wines or beer. Strong wines have an action similar to spirits, whereas weak wines more resemble beer.

In the case of wines, however, the stimulant effect of the alcohol which they contain is modified by the ethers which give to the wines their bouquet. It is probably in consequence of this that wines have a tendency to cause hilarity and an excitement of the emotions, feelings, and thoughts, whose effects are confined to the individual himself, rather than the tendency to movements of aggression upon others, which spirits have a tendency to produce.

The effect of alcohol in beer is modified by the hops. This substance has a somewhat soporific action, and beer has therefore a greater tendency to produce drowsiness and sleep than either spirits or wines.

The effect on the general nutrition of the body of wines and spirits is also different. The different appearance of the spirit and beer drinker has been illustrated by Hogarth in his "Beer Street" and "Gin Lane," where he depicts the beer drinker as large, fat, and bloated, the gin drinker as thin and emaciated. As a good example of the wine drinker, we may take the gouty old squire in the first picture of the "Marriage à la Mode." There we see him neither thin and emaciated nor fat and bloated, but with full, rounded, firm cheeks, smooth skin, and a general well-fed, well-cared for look. Unlike the gin drinker, he has no tendency to the accumulation of waste, *non-nitrogenous* products in the shape of fat. He, too, suffers, but his sufferings are due to the accumulation of nitrogenous products. Excess of uric acid and urates in his tissues render him irritable and choleric, while at the same time they cause gouty inflammation in his toes, which chains him to his chair like a prisoner, and occasionally subjects him to the tortures of the Inquisition.

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THREE CLINICAL LECTURES

ON

NERVOUS DISEASES IN CHILDREN.

Delivered at the Medical Graduates' College and Polyclinic.

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LECTURE I.

GENTLEMEN,—My object in giving this short course of lectures is to bring as succinctly and as clearly as possible before you the prominent features in connection with the commonest nervous diseases which affect children and young adults. I have included young adults in this list because it is very important, I think, to consider those in association with children, for in many, especially of the inherited diseases, one has to consider those diseases not only as they affect children—as they sometimes do—but also as they affect young adults. That is to say, the onset of such a disease occurs sometimes in very early life, but sometimes not until early adult life. Some of you may have seen the syllabus of lectures published in the 'Polyclinic,' and if I indulge in a little variation from the course there prescribed I must ask you to excuse me.

With regard to the paralytic diseases in children—and it is with those that I shall chiefly deal—there are certain differences which must strike one as being likely to occur in diseases in children, affecting mobility in them, as distinct from similar diseases which affect grown-up persons. In the first place, any disease which affects the nervous system of a child interferes with structures which are still immature and growing. By interfering with such structures the disease, of course, limits its effect upon the peripheral mechanism to which they are related, and in such a way it interferes

not only with the growth of the related limb but it also interferes with the function of the limb in a very striking way. Let me take as an illustration a case of hemiplegia occurring in an adult. In such a case we know that the limb or limbs which are affected have attained their maximum growth, and that the bones and other solid structures are not likely to be very much modified by any paralytic condition which ensues. On the other hand, if a child is affected with hemiplegia, we know that the nervous structures are still immature and growing, and that the limb or other peripheral structure related to the nervous structures is still small, and has not nearly attained the growth which it ultimately would have attained. Therefore, the disease process affects the nervous structure subserving the limb, and the growth and development of that limb are necessarily interfered with, sometimes, of course, in a very marked way, so that, *e. g.*, there is a great deal of shortening. But in every case affecting persons in early life there is always an interference with the development and growth of the peripheral structures related to the underlying nervous cells and fibres. Such interference with the growth of these structures is one point in which paralytic disease in childhood differs materially from a similar disease affecting a grown person.

Again, many nervous diseases in adults depend upon some vascular degeneration. For example, most cases of the disease we have already spoken of, hemiplegia in adults, are related either to atheroma or to some other condition affecting the vascular supply of the nervous system. In children, of course, the condition is totally different, except in those rare cases of syphilitic or renal disease in childhood which have associated with them changes in the walls of the vessels similar to those occurring in adults. But in the great majority of children the vessels are quite healthy, and the presence of degenerative changes, such as might lead to changes in the nerve-structures, is therefore very rare. So that in this way we should expect to have considerable differences in the mode of onset of the diseases causing such paralysis in children, and also probably in the nature of the process which underlies those diseases. We must also remember that in children and in young adults some of the paralytic diseases which have to be dealt with depend upon a curious

inherited tendency towards early decay which certain of the nerve-structures inherit. This tendency to early decay is one of those mysterious things the reason for which we do not know. Presumably it depends upon some condition of malnutrition affecting the young child, either while in the uterus or perhaps very soon after birth; but however it may arise it seems clear that in certain paralytic diseases in childhood and early adult life we have this tendency in certain structures or parts of the nervous system towards early decay. Possibly the most striking instance of such a condition, as regards cerebral diseases, is in some cases of what has been called diplegia, that is to say, a paralysis which affects both sides of the body. The name diplegia is usually limited to cases of that character which are due to some disease or defect in the intracranial structures on both sides. In some cases of diplegia there is a period of healthy life between birth and the time at which the diplegia or the paralytic symptoms of diplegia supervene.

As an example of this tendency towards early decay in certain structures in the spinal cord, we have to consider such diseases as Friedreich's ataxy. In that disease we have a condition of spinal cord in which certain parts of the spinal cord tend to decay very much earlier than they should do. This, as I say, is probably the result of some inherited tendency. What the nature or the ætiology of this inherited tendency is we have as yet no clear knowledge. Another class of instances of the inherited tendency to early decay is probably met with in cases which have recently been spoken of as muscular dystrophy. Cases of muscular dystrophy include cases of pseudo-hypertrophic paralysis, and cases of what used to be called idiopathic muscular atrophy. But I think it is much better to speak of them under the generic name of muscular dystrophy. Here, again, we have the same kind of predisposition to early decay and death in certain structures. In this instance the structures are not nervous, but they are so closely related to nervous structures, and they have such an intimate connection with the mobility of the part, that I think we are justified in considering them under the same category. So that in cases of diplegia, in which there is an interval of normal health between the birth of the child and the time of the onset of the paralysis, we have an example of the inherited tendency to early

decay in cerebral structures ; in Friedreich's ataxy and allied diseases we have an example of inherited tendency to decay in spinal structures, and in muscular dystrophy we have an example of inherited tendency to early decay in the muscular structures themselves.

Again, in considering the differences of disease affecting children, compared with disease occurring in adults, we have to remember that there are certain diseases which are almost peculiar to children. Probably the most common nervous disease occurring in childhood is the ordinary infantile paralysis, or anterior poliomyelitis. When I say this disease is almost peculiar to children I do so in the full knowledge that the same morbid process does occasionally, although comparatively rarely, affect adults. Still, for all ordinary purposes we may say that anterior poliomyelitis is a disease which is almost peculiar to children. We might similarly speak of post-diphtheritic paralysis ; and although post-diphtheritic paralysis does occur in adults, it is met with much more frequently, and in a much severer form, in children. The occurrence of these two diseases, with, at all events, markedly greater frequency and with markedly greater intensity in children, would seem to show that the young and growing structures which are present in childhood have a much greater tendency or liability to be severely affected by certain poisons than have similar structures in a grown person. Those, I think, are the chief points which we have to keep in mind in considering the differences in paralytic diseases as they affect children, compared with the diseases causing similar disabilities as they affect adults.

In considering the paralytic diseases affecting children and young adults, one has to regard them as they affect the different parts of the nervous system. We may, of course, have a disease affecting the brain. Of those diseases which involve the brain, the two most striking groups are, first, the group which I have already alluded to, that of diplegia, of which we have two divisions : those which occur at or before the time of birth, and secondly those which occur in the child after an interval of a few months or years of normal health.

Besides the diplegias we have another group, a very important one indeed, of paralytic diseases from affections of the brain in children, namely, cases of hemiplegia ; the condition as it occurs in the first few years of life, is attended at its onset with

special symptoms, and associated in its course with certain phenomena, about which I shall have more to say presently. Those are the chief diseases which affect the brain in childhood. No doubt, also, we have similar disease affecting the cerebellum ; we have conditions there probably of interference with the development of structures, and of a tendency to early decay in certain structures, although these groups of cerebral and cerebellar affections are rather ill-defined still, a result no doubt of the difficulty there so often is in getting the opportunity of examining children with these diseases after death. And besides these children very rarely do die actually of those diseases which have affected them in childhood, and then, after childhood is passed, the changes in the central nervous system are such as to give very little indication of the condition which was present at the time of the onset of the paralysis.

We have also to consider diseases of the spinal cord, those affecting first of all the grey matter. The chief example of that is anterior poliomyelitis, to which I have already alluded. There are also chronic diseases affecting the grey matter, similar to the progressive muscular atrophy in adults, not very common, it is true, but still such as may be considered in any course of lectures of this kind. Then, besides affections of the grey matter of the spinal cord, we have affections of the white matter of the cord, and those are chiefly of the variety I have spoken of, those due to diseases manifesting a tendency to early decay and death occurring in certain parts of the nervous system. The type of such disease is that which I have already mentioned, namely, Friedreich's ataxy. But there are also cases of spastic paralysis no doubt due to the same condition, cases of lateral sclerosis, as distinguished from the Friedreich's ataxy, in which the intensity of the degenerative changes falls upon the posterior columns.

We have also to consider, besides diseases of the brain and cerebellum and spinal cord, certain diseased conditions of the peripheral nerves which occur in children. In association with anterior poliomyelitis it was pointed out some years ago that there is frequently an affection of peripheral nerves, and I am inclined to think that occasionally we have an affection of peripheral nerves with similar onset to that of anterior poliomyelitis, but unassociated with any permanent affection of

the anterior horns, so that we may have a pure peripheral neuritis, probably the result of the same poison which causes the anterior horn affection. But besides that poison we know there is another poison which is very apt to cause peripheral neuritis, namely, alcohol, and although it is naturally rare in children, some cases have been recorded, and it is a condition which we must take into account, although we shall not have much to say about it. There is also another poison which we have heard a great deal of lately, a poison which affects children much more commonly, but by no means so frequently, namely, arsenic. A few years ago, when the treatment of chorea by large doses of liquor arsenicalis was much more fashionable than it is now, one saw far more cases of peripheral neuritis as a result of the arsenical treatment of chorea, and it was not at all uncommon to come across children, and even adults, who had been treated with large doses of arsenic, and who were, when they came under observation, suffering from typical arsenical neuritis. It is not common, but it is a condition which does occur, and it is one to which we should have our eyes open. The disease to which I have already alluded, diphtheria, is often followed by a condition of peripheral neuritis. I think it is very doubtful if the name given to that is a correct description of the actual disease; I question whether it is not better to look upon a diphtheritic paralysis as being the result of a toxic affection of the whole of the lower neuron affecting not only the peripheral nerves, but also affecting the central cell and causing degeneration, temporary it is true, of the whole of the lower motor neuron.

Besides those diseases of the spinal cord and brain and cerebellum, both grey matter and white matter and the peripheral nerves, we shall have also to speak of those conditions of muscular dystrophy to which I have alluded. These are a very important group of paralytic diseases, and they must be described. There are also a few peculiar diseases, more especially the diseases known as myasthenia and myotonia, or Thomsen's disease. And then there are the diseases known as myoclonus multiplex and paramyoclonus multiplex which should be alluded to.

Such is the course which I have mapped out, and upon which I should like to speak. As a system of lectures merely is not so useful as if it

is combined with something practical, I hope to show you cases illustrating as many forms of paralytic disease in children as I can contrive to get together.

I shall begin by speaking of cerebral paralyses, those which occur in children and young adults as the result of cerebral affection. First of all I will speak of diplegias. The name diplegia is really one which means a paralysis of both sides of the body, but in the ordinary nomenclature it has come to be restricted, as a matter of custom, to cases of that character which are due to cerebral disease. As I have said, there are two forms of diplegia which one has to consider. First of all, there is the diplegia which occurs at the time of birth. When a child who is affected with this condition is seen, it may be said that the history is usually that the child was late in walking. Generally it has not walked until it has reached the age of four or five years, and then its gait has been a peculiar one. Usually it progresses with very stiff legs and with a spastic walk, that which is known as the cross-legged gait. The explanation is that there is some very strong adductor spasm on both sides, so that one leg in being brought forward is crossed a little in order to get it in front of the other. Such a child is characterised by this peculiar cross-legged gait, and the reflexes are, as a rule, exaggerated, and ankle-clonus may be present. In many cases the legs are the only part affected, and such cases are spoken of as cases of cerebral spastic paralysis. The arms in these patients are often quite useful, and the patient is able to carry out ordinary movements with the arms just as well as any other person is. But even in such cases I think you can often find, on going into the history, that the child has been late in acquiring the power of using his arms freely, and that there has been some slight affection of the arms at first, even in cases in which the paralysis is afterwards limited to the legs. In other cases you find that not only are the legs affected, but that one arm is affected in a similar spastic way to that in a case of hemiplegia. But whereas in a case of hemiplegia one leg only is affected, in the condition I have referred to both legs are affected and one arm, or in another group of cases you may find both arms as well as both legs are involved, so that the child finds very great difficulty in using its arms with accuracy. When

the arms and legs are both affected, which is rare, the face may escape, or the face may be affected in an unequal way, though not in such a marked way as the limbs. In such a case you not infrequently have squinting present, as if the ocular movements had suffered from the paralysis. With regard to the spastic condition especially, certain cases have, associated with paralysis, irregular movements. Such movements are sometimes spoken of as choreiform, and they do sometimes closely resemble, or at all events strongly suggest, the movements of chorea. But in other cases the movements, although very distinct and striking, are scarcely described accurately as having the character of the movements present in chorea. The name athetosis has been used in connection with such movements, and undoubtedly the athetoid character of these movements is fairly common; in those cases especially in which both arms and both legs are involved you may have athetosis present on both sides, and it is then spoken of as a double athetosis. Occasionally when one side is affected you may have athetosis on that side, but true athetosis is much more common in the hemiplegic form of cerebral paralysis, of which I shall speak presently. But it does occur in diplegia, and its presence in association with that condition ought to be recognised.

So much for the physical symptoms. Every now and again such a patient is the subject of fits, and when the ætiology and the pathology are considered in connection with the disease, I think what will strike one with surprise is not that fits are occasionally present, but that they are not more common in such a condition than they actually are found to be. When we recognise that the condition is the result of some cerebral affection, it will scarcely be wondered at that there are frequently associated with the physical symptoms also mental alterations. The mental alterations are rather uncommon, in my experience, in the cases in which the legs only are affected. In the latter case it is not unusual for the patient to have a very good mental condition indeed, and some of the children of this description whom I have seen have been almost unnaturally sharp. But when the four limbs are affected there is a very strong probability that mental alterations are also present, and sometimes they are very profound. As regards the cases in which the legs

only are affected, the child is nearly always late in walking, and always walks imperfectly. In reference to the others, it may be said that it is very rarely they are able to walk at all, and when they do walk it is only with very great difficulty, and with a considerable degree of abnormality.

We next come to ask what is the ætiology of such cases? You find in the great majority of such cases, I believe, that there is a history of some difficulty in connection with the entrance of the child into the world, that there has been a difficulty in connection with the labour. It is very striking that so many of the children who suffer in this way are the first-born children of their parents. Another point is that the state not unusually affects the first-born children of somewhat elderly primiparæ, and, as I have said before, the labour is usually a long one, the presentation often abnormal, and not infrequently the labour is found to have been an instrumental one. Such facts as those are fairly striking. In thirty-two such cases which I have had under observation, and in which I have obtained a reliable history, the children have been, in fifteen of the instances, the first-born children, and in these as well as in ten more, that is to say, in twenty-five out of the thirty-two, there has been a difficulty in the labour, the labour has been an unusually long labour, and has frequently been an instrumental one. In a few cases, it is true, there is a history also of another abnormality in the labour, viz. that it has been a precipitate labour; the child has been born very quickly, and the mother has been unassisted at the time the child was born; sometimes the event has come on so quickly that not even the nurse has been present. As I say, these facts all suggest some actual lesion occurring in connection with birth as accounting for this paralysis, and there has been found in new-born children hæmorrhage over the vertex, and in some cases hæmorrhage over the base, and it is suggested (and I must say that the suggestion seems to me to carry a great amount of force) that in at least some of those cases of diplegia the paralysis is the result of actual hæmorrhage over the surface of the cortex, or, perhaps sometimes of brain laceration, and that such lesions result, not as some people have said, from instruments used to effect delivery, but rather from the conditions which so frequently render the use of instruments necessary.

If we consider the peculiar relation of the different areas of the cortex over the hemispheres to the middle line, we can see how the symptoms may be accounted for. Nearest the middle line are the leg areas on each side, next these the arm areas, and still lower those for the face, and further forward there are the centres controlling the movements of the eyes, in the prefrontal lobes. You can see how hæmorrhage occurring to a different extent would account for the curious variety of cases which we have. Suppose we have a hæmorrhage occurring on each side of the longitudinal fissure, and of small extent, such a hæmorrhage would affect the leg areas, and give rise to a form of cerebral spastic paralysis which is so common, that form of diplegia known as cerebral spastic paralysis, in which the condition is limited to an affection of both lower limbs. In such a case the patient walks with a spastic gait, but mentally and physically otherwise the child appears to be quite well. In some cases we have an affection not only of both legs but also of one arm. We can quite understand the occurrence of such a condition if we imagine this hæmorrhage extending on one side, so as to catch in the arm area as well. I have mentioned that there are cases in which there is not only an affection of both the legs and one arm, but of both legs and both arms, and I have said that in such cases the face is often affected too. If we suppose such a hæmorrhage as I have depicted spreading still further, so as to catch in the leg and arm area on each side, we have a condition giving rise to paralysis in both the arms and both legs, and you can see how it might affect the face if it were still more extensive. To account for squinting one must picture the hæmorrhage spreading forwards and catching in the areas which have to do with the movements of the eyes, the most anterior of all the motor areas. And if we suppose that the hæmorrhage spreads still further forwards, that is to say, in those cases where the hæmorrhage is very extensive, we should have a condition affecting the prefrontal lobes which might account for any degree, even a severe degree, of mental impairment. So, although we have no definite proof post mortem of this condition as actually occurring in well-established cases of birth palsy or diplegia, we have post-mortem evidence of hæmorrhage occurring on each side of the longitudinal fissure

in newly-born children, and the presumption from the consideration of clinical and pathological facts, I think, is quite justifiable that such a morbid anatomy is the most likely one in the majority of these cases. It seems to me that the mechanical difficulties associated with labour in so many cases, and the fact that it is a first labour, and so often an instrumental labour, seems to point strongly to the occurrence of some traumatic affection as the cause of the diplegia. And when we have, as I say, actual evidences, in some cases, of hæmorrhage occurring in new-born children who have been born in this way, I think we may assume that in at least many of the cases this morbid anatomy is the most likely one. So much for the morbid anatomy and ætiology and pathology.

Let us now say a little about the diagnosis. The diagnosis is only really difficult in those cases in which the affection is limited to the legs, and the condition which would be likely to be mistaken for it would be some affection of the spinal cord, giving rise to similar symptoms. Probably the most common affection of the spinal cord in children which would cause similar symptoms is pressure by caries of the spine causing an affection of the lateral columns, and the prominent clinical symptom in such a case is a condition of spastic paraplegia, an affection of both legs, as in diplegia. In such a case one must rely, first of all, on the history of the condition. In diplegia such a condition will be present from the birth of the child; the subject of the trouble will never have walked well. In spinal caries there will have been an interval of time, during which the child has been able to walk perfectly well, and probably there will also be present some deformity of the spinal column itself, some angular curvature. Those two points, the history showing the period during which the child was able to walk quite well and the presence or absence of actual angular curvature of the spine, will determine whether it is a case of spinal caries or diplegia.

With regard to prognosis, one can always be very hopeful in those cases, to the extent of prophesying certain improvement. It is difficult in the early stage to say what the degree of improvement which will take place will actually be; but in the great majority of cases, especially if there is no evidence of affection of the upper limbs, it may be definitely stated that the child

will be able to walk, but the parents must also be warned that the child will never be able to walk normally, and will probably always walk with difficulty.

With regard to cases in which both upper and lower limbs are affected, and more especially when a certain amount of mental defect is present, one must be guarded in prognosis. Such children generally remain weak-minded, and although there may be some physical improvement the child is never really well.

With regard to treatment, can one do anything by treatment to get the child well or improve its condition? Undoubtedly when one sees the patient early one has to see chiefly that deformities are not allowed to develop. There is a strong tendency for contractures to take place, especially at the knee, and in very early life it is difficult to get the legs straight and in line with the thigh. There is also a tendency for the ham-strings to be contracted. Therefore every effort must be made to get those structures into good condition and prevent the ham-strings or other muscles from contracting, and so making walking more difficult, and also towards preventing malposition and so on, which would increase the difficulty the child would have in getting about. So what has to be done in the treatment of a child suffering in this way is to see that every chance is given to it to develop in a normal manner, to take every possible measure to prevent contractures (*e. g.* massage and passive movements), and if one has seen a child when contracture has taken place it should be a matter of consideration whether the child might not be helped by division of the tendons or by the provision of a light form of apparatus which would make walking easier. Unfortunately nothing can be done for the mental condition, except with regard to measures which can be taken by means of careful discipline and education. And such cases are very much better treated in some asylum or other institution where their potentialities and limitations are better understood. When fits are present they must be treated in the ordinary way, and as far as my experience tells me those fits, in association with organic disease of this kind, are even more amenable to treatment by means of bromides than are the fits of ordinary epilepsy; at all events they are, as a rule, easily controlled by bromides in comparatively small doses. So

much for the diplegias which occur at the time of birth.

I now wish to say a few words about diplegia which occurs in an individual of comparatively normal development. It has been recently shown that such cases are not at all uncommon; they are usually characterised by the onset of the disease after some more or less definitely described acute illness. There seems, however, in a good many of the cases, to be grave doubt whether there is any very acute illness preceding the onset of the paralytic symptoms. The condition is very rapidly characterised by the presence of rigidity, of head retraction, and of nystagmus. Sometimes the child becomes blind, and then the condition is one of extreme spastic rigidity of the whole body. Such cases have been actually examined after death, and, of course, they nearly always do die. The condition found post mortem has been one in which the pyramidal cells have been very much interfered with in their development; there is evidence of very general decay and destruction of the pyramidal cells, and those are cases in which, as I have said, there seems reason to suppose that the child has inherited a nervous system in which there are certain structures which are prone to early death. Also there is a certain group of cases which have been described by Mr. Waren Tay first of all, and more recently other cases of the same group have been described and carefully investigated by Dr. Risien Russell and Mr. Kingdon, and I believe all the recorded cases are in Jewish children, and they are characterised by the gradual loss of power and wasting, and also by curious ocular changes, optic atrophy associated with changes in the macula lutea, and in those cases there is found to be also marked degeneration of the pyramidal cells of the cortex and related structures, and degeneration of the cells of the retina. Those cases are rare, but they ought to be mentioned because they are cases of diplegia which occur in children and occur in contradistinction to the cases of birth palsy. This latter class of cases occurs in children after an interval of apparently normal health.

In speaking of infantile hemiplegia we should exclude from consideration hemiplegia occurring in acute illnesses and due to the same causes as are present in adult life, cases of embolism occurring in the course of scarlet fever and rheumatic fever,

and cases of hemiplegia also occurring in the course of diphtheria. Infantile hemiplegia proper is the hemiplegia which occurs in the first five or six years of life in a child who had been previously perfectly healthy. The mode of onset is with acute illness and high temperature, with a convulsion, or a series of convulsions, affecting one side, the side which afterwards becomes paralysed. After the occurrence of the paralysis there may be a continuance of the fits, and the child may be extremely ill, and, of course, sometimes the illness ends fatally. However, when the child comes under observation it is found to be paralysed in the arm or leg, and usually also the face is slightly affected. The face is not so much affected as in the adult, but the arm and the leg are very similarly affected. There is very rarely any affection of sensation, and very rarely, at all events when the patient comes under observation, is there any hemianopia. It is said that hemianopia and hemianæsthesia are both symptoms not infrequently present at the time of the onset of the condition. Another thing which strikes one, but only after the lapse of some time from the onset, besides the hemiplegic condition of the child, is the maldevelopment of the affected side. No doubt in this maldevelopment—or atrophy, as it is sometimes improperly called—there is more than merely the effect of disuse, and it seems as if the structures which are subserving the affected limbs have been so much interfered with that they cease to have the proper nutritional effect upon the corresponding limb. So that a child who has hemiplegia, within a few months after the onset of the paralysis shows nearly always a striking difference between the affected limbs and those of the opposite side. I have spoken of the persistence of fits, but sometimes they cease altogether; but when they do persist they are not uncommonly of a severe character, affecting either the one or the other side, and sometimes spreading to both sides, although sometimes they are of a very slight character. We occasionally find slight fits followed by that peculiar condition of post-epileptic automatism which we find after the attacks of *petit mal* of true epilepsy. The associated spontaneous movements in infantile hemiplegia are very striking, and they probably constitute the most marked difference between those cases and adult hemiplegia. Athetosis, or the mobile spasm which occurs in infantile hemiplegia, is a rare accompani-

ment of hemiplegia in the adult, and if you see an adult with hemiplegia and athetosis, you may be certain, in the great majority of cases, that the hemiplegia of that adult dates from very early in life.

As regards the diagnosis, that is not difficult; you have a hemiplegic condition and you nearly always have the history quite clearly, and the condition is not likely to be confounded with any other. I might mention first of all the pathology. That has been ascribed to various causes. There is little doubt that the disease is a toxic one, because it occurs frequently in association with what is fairly recognised as a toxic disease, namely, anterior poliomyelitis. I have known one instance of a patient in the same family attacked with anterior poliomyelitis, another member of the same family having infantile hemiplegia, and the presumption is that this poison finds its way into the nervous system through the blood-vessels, and that clotting takes place in the blood-vessels, either in the veins or in the arteries, we do not know which. It is presumed that as a consequence of this we get the disturbance in the nervous system which follows. Some have said that the condition is one of acute inflammation of the cortical cells, and such a condition has been described in the adult. But, as far as I am aware, no definite evidence of the pathology has been brought forward affecting children.

The treatment divides itself naturally into two parts, namely, the course to be pursued when you see a case of this kind at the onset, and secondly the treatment which you follow when the condition is established. The treatment at the time of onset resolves itself into attention to the convulsions, as they are a striking feature, and if you are controlling the convulsions you are doing all you can for the child. The first step is to give a large dose of calomel, two or three grains, and before this has acted, if the convulsions are a severe and prominent feature, it is well to give a large dose of bromide, and such a dose is best given by injection; it may be either half a drachm or a drachm, injected into the rectum. If the convulsions are slight or infrequent, fifteen to twenty grains would be a much better dose. But when you have to deal with a convulsion as one of the prominent symptoms of the illness, it is better to give a fairly large dose of bromide, and to give it

by the bowel, because when it is given by the stomach in such a condition it is often not properly absorbed. The acuteness of the onset having subsided, we have to deal with the prominent condition remaining, and we must unfortunately recognise that the paralysis is not likely to pass away completely. Great care must be taken in this, as in the previous class of cases, to prevent contracture and deformities. The tendency is for contracture to take place in the ham-strings, and also in other muscles, more especially of the lower limbs, and every care must be taken to avoid that; and, if it has taken place, the proper measures must be taken to rectify it by passive movements and stretching, or by actual tenotomy. Sometimes it will be necessary to give a slight support to the child because of the inversion and deformity which are apt to take place in the foot. Such an instrument will often make walking much easier. Another important feature is the treatment of the fits; and those, again, are very amenable to the action of bromides. In a great many of the cases you will find that the fits yield readily to bromides, although occasionally one meets with a case in which bromides seem to be of no use. The mental condition of the patient has also to be considered. In a certain number of these cases of illness the patient is undoubtedly mentally affected. Often that mental involvement is slight, but sometimes it is severe. Usually we have to deal with a certain degree of mental peculiarity, and we should always have our eyes open to the possibility of such a condition in any case seen at its onset. Sometimes the mental condition is one which is very bad, and the patient has to be sent to some institution; but as a rule the mental state, although peculiar and a little unstable, is not so depraved as to necessitate such a measure.

In a long discussion in the 'Berliner klin. Woch.,' December 16th, 1901, Kuttner states that sounds of splashing in the stomach are pathological and show a loss of tone in the muscles of the stomach and abdominal wall. The position of the stomach is secondary. If it is heard during digestion, gastric atony is present; if heard a long time after meals, there is motor insufficiency of the stomach. Following gastric atony, gastroptosis may develop. —*Philadelphia Med. Journ.*, February 8th.

THE MIDWIFERY OF THE FUTURE.*

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GENTLEMEN,—I purpose to indicate in what ways the practice of midwifery is likely to be altered in the future, and ought to be altered. I say not that the time is ripe for a universal adoption of the changes I specify; much education, both of the profession and the public, is required. But I am convinced that if the profession and the public could adopt the views that I shall put forward, it would be good for both.

When you go to attend a woman during childbirth what are you going to do? Childbirth is a natural process. In all the lower animals and among savages it is finished without help; why, then, cannot civilised women have babies without a doctor's presence? All that the doctor does in an absolutely normal case is to tie and cut the cord and to press the placenta out of the vagina, and it requires no very high education to do these things. He speaks to the patient, tells her everything is well, and thus removes or lessens fear; the value of this depends on the confidence the patient has in him, not on his obstetric skill. He helps the nurse in making the patient clean, dry, and comfortable afterwards. Among the higher classes he also acts as an anæsthetist, but neither anæsthetising nor helping the nurse is properly obstetrical work.

If you asked an accoucheur of 100 years ago what he was with a woman in childbirth for, he would tell you it was to give assistance if the child was not advancing properly into the world. If you pressed for details you would find that his idea of proper delivery was partly conditioned by time and partly by the state of the patient. If within a certain fixed period after the dilatation of the os the child was not delivered, or if the patient showed signs of great physical exhaustion (and in their appreciation of the signs of exhaustion I think the older accoucheurs were as correct as many of the moderns), then the accoucheur pulled at its head with forceps, or turned it and pulled at its leg; if he did not succeed, he perforated the child's head and then delivered. His

* Delivered before the London Hospital Medical Society.

view was that it was his business to watch what was going on, and help, either by pulling or by lessening the size of the child, if the power of the uterus failed.

Within my memory occurred the publication of a case occurring in a London hospital, and attended by the obstetric physician to the hospital, in which, as delivery did not take place, first of all pulling with forceps was tried; this was fruitless, so then the head was perforated. When this had been done, still the physician could not pull the child through the pelvis, so he performed Cæsarean section; and finally the woman died. This was a case of bad practice from beginning to end.

The better way—which ought to be, and will be, the practice of the future—is to find out mechanical difficulty beforehand.

With a pelvis of average size, a child of average size, healthy passages, and a normal presentation, there can be no mechanical hindrance to natural delivery. When the head is in the first position the longest diameter occupying the pelvis is the occipito-frontal; this measures about four inches and a half, and it lies in the oblique diameter of the pelvis, which measures about five inches. In the conjugate diameter, which is usually about four inches and a quarter, lies a diameter running from behind the right parietal eminence to in front of the left one, and measuring about four inches. Thus nowhere is there any hindrance to the passage of the head through the brim. Below the brim the pelvic space enlarges. As the head descends it becomes flexed, so that instead of the occipito-frontal measuring four inches and a half, the sub-occipito-frontal, which is only four inches, occupies first the oblique, and then the antero-posterior diameter of the pelvis. A foetal head of average size can be put through a pelvis of average size without touching the bones. As the head is the largest and hardest part of the fœtus, where it has passed the trunk can follow.

In such a case, therefore, there is not, and cannot arise, any hindrance to delivery from the pelvic bones. If labour is long it can only be because the soft parts dilate slowly. The whole business of normal labour is the stretching open of the soft parts.

If the genital passage is healthy it will always in time open up. Whether it stretches open quickly

or slowly depends upon the power with which the child is pressed into it; in other words, on whether the pains are strong or weak. Weak pains will make the labour long, but they bring with them no sort of risk. A pathological condition has been described, or rather talked about, called "missed labour," in which it is supposed that labour either comes not on or is not completed. Such a thing is unknown in a woman having a healthy uterus with a living child inside it. The cases reported under this title were cases either of ectopic pregnancy, cases of retention of a dead child *in utero*, or cases in which delivery was hindered by disease of the uterus. When labour is natural (meaning by this that the pelvis is normal, the child normal, and the genital canal healthy) delivery will always take place, and there is not, and never can be, any necessity for interfering.

Delivery may be hastened by pulling at the child, and it may be that the more severe pain caused by quickly stretching open the passage may affect the patient's nervous tone less than the multiplication of less severe pains involved in the more gradual stretching conducted by Nature. If the patient is anæsthetised, hurrying delivery abridges the time during which the patient is under the anæsthetic, and therefore the amount of it that she will take; it also is a considerable economy of the accoucheur's time. In these ways the artificial hastening of delivery may be a good thing, but it is not necessary. It is possible to do damage by pulling too violently, at the wrong time, and with badly applied instruments. The accoucheur is under constant temptation to hurry on delivery because his time is precious, and if he yields to this temptation the cervix and vagina may be cut, the perineum may be ruptured, veins in the child's pia mater torn, causing death by meningeal hæmorrhage, and if the child is dragged out while the uterus is passive, post-partum hæmorrhage will probably follow.

Interference in natural labour is never necessary, and may do much harm.

What then, in natural labour, has a doctor got to do? The patient may wish for anæsthesia, but acting as an anæsthetist is not an obstetrical service, although the obstetrician may do it. Obstetrically, all that the doctor has to do in natural labour is to watch the parts dilate and the child emerge, then to tie and divide the cord.

and, some half-hour or so after, to press the placenta out of the vagina.

Does it need a highly educated man to do this? I think that the average woman could do these simple things as well as the most highly trained doctor, and I think it a pure waste of time for a doctor to sit by watching the passage dilate in a case of natural labour.

It may be said that the doctor is there to supervise the nurse. It is much to be wished that patients would be guided by the doctor in choosing a nurse. The doctor ought to satisfy himself that the nurse understands her business before he sanctions the patient's choice. But this inquiry ought not to be postponed till the time of labour.

At present the notion is rooted in the female mind that a child cannot be safely brought forth unless a doctor is in the room. So long as this is so doctors will have to waste their time in watching the genital passage dilate, tying umbilical cords, and pressing out placenta.

The first thing needed for improvement in midwifery practice is to educate the public up to the belief that difficult labour ought not to occur, for it can be prevented. In order to prevent it, medical men should instruct their patients to come for examination before the end of the eighth month of pregnancy. In the case of first pregnancies it would be well to do so at the end of the seventh month.

The object of this examination is to ascertain four things:—(1) The size of the pelvis; (2) the size of the child; (3) the relative size of the child and the pelvis; (4) the position of the child.

The size of the pelvis is important, not only for the delivery which is expected in a month or two, but for all future labours. If the pelvis is small the probability is that in most of the patient's pregnancies something will have to be done to prevent difficulty in delivery. If it is large, the probability is that future labours will be easy. But so far as the presently expected labour is concerned the size of the pelvis is less important than the relative size of pelvis and child.

The external measurements, the inter-spinous, inter-cristal, and external conjugate, should be taken during pregnancy. They are unimportant unless they are much below the average, but the taking them inflicts no pain and is in no way detri-

mental to the patient, and therefore there is no harm in taking them, and now and then they may give useful guidance.

The internal measurement of the diagonal conjugate is more important; but if the pelvis is of normal size you will not be able to reach the sacral promontory without an amount of upward pressure that will be extremely disagreeable to the patient. Routine attempts to do this would probably lead to considerable diminution of your opportunities of doing it. It is better, therefore, to be content with finding out that you cannot reach the promontory without hurting the patient, and conclude from this that there is no great pelvic contraction.

After a first labour the true conjugate should be measured by introducing the hand during the third stage of labour, after the method described by Robert Wallace Johnson. Any other measurement which is shortened to four inches or less should be measured in the same way. This involves a very little addition to the unpleasant manipulations of the third stage of labour. If the patient is anaesthetised it involves none at all, and if not anaesthetised the patient will take it as an inevitable part of a disagreeable business. When this measurement is known you are in sure possession of one of the factors upon which the ease or difficulty of delivery depends.

If we know the size of the pelvis we want next to know the size of the foetus and its head. If the foetus is a healthy one, its head will bear the usual relation to the body. We cannot measure the foetus directly, for it is contained in the uterus, and the uterus is contained in the abdomen. The size of the uterus is conditioned not only by that of the foetus, but by the amount of the liquor amnii, and the size of the abdomen depends not alone on that of the uterus, but on the bony and muscular development of the woman, the size of her pelvis, of her chest, and the amount of fat. These are reasons which make it impossible to always infer the size of the child from the measurement of the mother's abdomen.

But although there are variations in the measurements of the abdomen not due to the size of the foetus *in utero*, yet there is a mean in these measurements, and the patients who are near the mean are much more numerous than those who are far from it, and patients of the latter class are not difficult to recognise. It needs no very acute

perception to notice that a woman is broader and bigger and fatter than most women.

Women who are bearing children are not generally very fat. The average greatest girth of a woman of medium size at the full term of pregnancy is under thirty-six inches—or one yard. The average measurement in such a patient—from the symphysis pubis, over the convexity of the uterus, to the highest point of the fundus—is thirteen inches. If these measurements are not exceeded the child is not of more than average size, and if we know the pelvis to be of normal size we may predict a safe and easy delivery so far as mechanical conditions are concerned.

If these measurements are much exceeded it is possible that the increase may be due to a condition such as a tumour, dropsy, or excessive size of the child, which it is extremely important to detect early; or it may simply be that the woman is big and fat, although the child is normal in size. In a first labour we may not accurately know the size of the pelvis. If, then, we know not exactly the size of either of the two factors upon which the mechanical problem of labour depends, we then must proceed to find out their relative size. If the child is big it matters not if the pelvis is big also, and a woman with an average-sized child but a small pelvis may have a difficult labour. The thing essential to be known, if we are to forecast the mechanical ease or difficulty of labour, is the relative size of the pelvis and the foetal head. The absolute measurements are only useful because they help us in determining this—the main thing to be found out.

The necessary preliminary to ascertaining the relative size of the head and the pelvis is to see that the head is over or in the pelvic brim, to find out the position of the child, and, if the head is not presenting, to turn the child by external palpation so that the head may present. The possibility of doing this is one of the advantages of examining the patient during pregnancy, instead of waiting till she has been some time in labour before you have the opportunity of finding out that all is not right. But as the head presents in 95 per cent. of cases, the patients who will benefit by having the position of the child altered will be few.

Having ascertained that the head is presenting, or made it present, then see that the child's back is

in front, and if it is not, turn the child round (by pressing its shoulders in opposite directions) until its back is in front. Then put the patient in a reclining position half-way between sitting and lying. In this position the long axis of the uterus will be vertical, and nearly at right angles to the axis of the brim. If, as ought to be the case at eight months' pregnancy, the head is small in relation to the pelvis, it will, by its own weight, sink into the brim. If it does not, then apply the hands, one on each side, with the finger-tips opposite the neck, and then, with the points of the fingers pressing on each end of the foetal head, press it down into the brim. If the condition of things is normal you can easily do this.

Suppose, then, that you have a patient about to have a child. You have examined her when she was eight months pregnant, and again at nine months. You know she has no pelvic deformity. You know that the child is not of excessive size. The inference from these facts is that the child will easily enter the pelvis, and you have verified that inference by examination. Is there any need for you to sit by her side during labour? Are you the only person who can tie the cord? Will the patient come to harm if you are not the person who presses out the placenta?

If the patient wishes an anæsthetic, of course she must have a doctor with her. But suppose she is a poor patient, to whom the amount of the fee is a serious matter. Such a patient, *after the doctor has found out that all is right*, will be just as safe with a competent midwife as with a doctor. If the pains are weak and the labour long, the patient will come to no harm from that. The child will certainly be born sooner or later, and if the uterus is let alone it will expel the placenta and afterwards contract and retract. If the patient should have the misfortune to be attended by a doctor in a hurry, who will not wait for the uterus to dilate the passage and expel the child, but must needs seize the child and pull it out as soon as he can persuade the patient to let him, and, when he has accomplished this, then squeeze out the placenta with similar haste, she will probably be an example illustrative of Professor Sinclair's address at Montreal.

We hear a great deal now about midwives. The practice of midwives, I fear, is bad, for in spite of the abundant evidence afforded by lying-

in hospitals that puerperal fever can be prevented, in this country it has not yet appreciably diminished. This shows that women are largely attended in their confinements by persons who understand not the use of antiseptics, and the proportion of midwives and monthly nurses who are thus ignorant is probably larger than the proportion of doctors. But there are agencies at work which are diminishing this evil. Elderly medical men who have attended thousands of cases without antiseptics, and therefore believe not in them, are yearly dying out, and being replaced by young men trained to look upon Lord Lister's work as the foundation of successful surgery. A similar statement cannot be made about midwives, for it is still possible for any ignorant woman, with dirty hands and a dirtier dress, to call herself a midwife, and poison her patients. But there is a yearly influx, from our different lying-in hospitals, of younger women who have been taught antiseptics, and the knowledge on this important subject must gradually extend, not only to those who call themselves midwives, but also to the public, so that I think this is an evil which is slowly being ameliorated.

It is common in poor neighbourhoods for midwives to co-operate with medical men, and as a rule such co-operation is carried on in the worst possible way. The midwife is called to the case; if she finds a transverse presentation she is supposed to send for the doctor, but she probably, and not unwisely, turns. If there is hæmorrhage she will send, but if there is a head presentation she waits and watches; if the patient gets in a dangerous state she sends for the doctor. The patient is allowed to drift into danger through the midwife's ignorance, and when the doctor is sent for it is generally too late for him to be of use.

Now I urge that it is not the best use to make of the doctor to send for him because a midwife has mismanaged the case; the doctor cannot do the best thing for the patient unless he sees the case early.

I suggest that a doctor who practises in a neighbourhood such that he is obliged to attend a number of poor women, should employ midwives. When poor women, who cannot afford his fee for acting as an anæsthetist, come to ask his attendance in childbirth, he should explain to them the far greater benefit of preventing difficulty than of

treating it. He should insist on the patient coming to him for examination when she is eight months pregnant, and again when she has reached full term. If at the latter period he is sure that the child is not of excessive size, that the pelvis is normal, and the head engaged in it, then he should explain to the patient that she will, in all probability, be delivered naturally, and that the midwife can do all that is required after the child is born: that the patient should send for him immediately she is taken in labour that he may ascertain that everything is still normal (for the child may have changed its position), but that if it is going to be a normal delivery she is not to expect him to sit by and watch it. If this were done it would be a great economy of the doctor's time, the midwife would have a more intelligent conception of her task, and the patients would be protected against bad practice, for difficulty in delivery would be prevented instead of being treated.

In the foregoing remarks I have assumed that the case is one in which normal delivery is to take place. They apply to at least 90 per cent. of labour, and therefore to the great bulk of the work of any practitioner.

But what about the remaining 5 or 10 per cent. of labours? By insisting that the patient should come for examination not later than the eighth month of pregnancy, nineteen out of twenty difficult labours could be prevented. Take first the cases in which the pelvis is normal in size. What causes can produce difficulty here? There are but two—excessive size of the child and malpositions. If at eight months pregnancy measurements show that the child is as large or larger than it ought to be at full term, the induction of premature labour will obviate difficulty, and is clearly indicated. The induction of premature labour, when the pelvis is normal, because the child, although the pregnancy has not reached full term, is as large as it ought to be at term, is not likely to be attended with the disappointment of a child born too feeble to survive.

Consider the case of pelvic contraction. If the contraction is slight, by repeated examinations during the last two months of pregnancy, the time can be perceived at which the head begins to have difficulty in sinking into the pelvic brim, and then is the time to induce labour. An induced labour, if properly done, ought to be no more dangerous

than a normal labour. From the point of view of the mother, it is better to err on the side of inducing labour too soon rather than of waiting too long.

The objection to premature delivery is the risk to the child. The mother may be anxious to have a strong vigorous baby, and her pelvis may be so small that the child will only pass through it if delivered at such an intra-uterine age that there will be unusual difficulty in bringing it up. In such case the proper course is to advise Cæsarean section.

There are two things which are peculiar to Cæsarean section:—(1) It is the simplest and easiest, and therefore ought to be the safest of all abdominal operations. There is a proverb, which applies to most abdominal operations, to the effect that you never can tell what is inside a box until you have got the lid off; this applies not to Cæsarean section. Here you know exactly what is inside the box; you are dealing, not with disease, but with healthy parts; you will find each structure easily recognisable, in its proper situation, not distorted or displaced; there are none of the adhesions which so often give trouble in other operations; the different steps of the operation can be thought out and planned beforehand, with a practical certainty that nothing unexpected can be met with. It has no necessary mortality; the causes which have so often led to death in times past can now be prevented. Hæmorrhage and peritonitis are practically the only dangers; peritonitis can be prevented by cleanliness and antiseptics; hæmorrhage can be prevented by a proper method of suture. The other peculiarity of Cæsarean section is this—that there is no other operation which displays so clearly the influence of the state of the patient on the result. Cæsarean section, most safe when performed upon a healthy subject, is most deadly when the patient is exhausted by protracted labour. In this hospital during the last twenty years the total mortality from the operation has been nearly half, but of the cases in which the operation was performed before the onset of labour not one has died. The mortality has been due to the cases taken into the hospital after they had been in labour many hours outside.

Cæsarian section has this advantage—that it can be followed, if wished, by sterilisation of the

patient. This can be done or not, according to the wish of the patient. The best way of doing it is to remove the body of the uterus, leaving the ovaries, or one ovary, behind. Tying the Fallopian tubes does not prevent conception. The uterus has to be sewn up in some way, and there is no greater risk in sewing up the cervix, after the body of the uterus has been cut off, than in sewing up the body. If the patient is not to become pregnant again the body of the uterus is an organ functionally useless, the cause of periodical ill-health, and, therefore, better removed.

The induction of premature labour has hitherto been the usual advice given to the possessors of small pelves, and the date has been arbitrarily fixed according to the size of the pelvis; but the proper date cannot be fixed in this simple manner. The determination of the relative size of the head and the pelvis is the right guide to date. But, passing this point, is the induction of premature labour, because the pelvis is contracted, so very satisfactory a solution of the difficulty? The smaller the pelvis the earlier must labour be induced, the smaller will be the child, and the greater the difficulty of rearing it. The prospect of ten or twelve labours, with perhaps two or three children only surviving, is not a very satisfactory one. When the safety of Cæsarean section becomes recognised I think it will be judged more satisfactory to all parties to deliver a strong full-sized child at full term, by the abdominal operation, as many times as the patient wishes, and, when she is satisfied with her fertility, to complete the operation by removing the body of the uterus.

In some cases, symphysiotomy will enable a living child to be born through a pelvis which otherwise would not allow it to pass; but this operation is only useful in a certain definite relation of child and pelvis. If the child is too large to be delivered through the pelvis when enlarged by separation of the pubic bones to the extent of two inches, and yet the operation is done and the child dragged through, there is great danger of serious injury to the urethra and bladder; hence, before symphysiotomy is done, the relative size of child and pelvis must be most carefully determined. In Cæsarean section, the necessity for operative delivery of some kind being granted, the size of the child and the pelvis are both immaterial.

Your patient may have a transverse position of

the foetus. A general proposition applies to all transverse positions, without exception, viz. that the earlier the position is recognised the easier it is to treat it. If your patient sends at the first indication that labour is beginning, and you get to her before the membranes are ruptured, it is easy to alter the position of the child; if you are not summoned till the patient has been long in labour it may be very difficult.

If you get to the case early, you know the pelvis to be of full size, and you judge the child not to be larger than usual, you will probably be able to bring about a head presentation, and then the midwife can be left to watch the birth of the child just as if it had all along been lying with the head in the brim.

If a head presentation cannot be brought about the attendance of a skilful person is necessary if the child is to have the best chance of being born alive. So far as the mother is concerned, when the child has been put so that its long axis lies in the genital canal, and no cause of obstruction is present, she is safe, and the midwife can do all that is required as well as the doctor; but there is danger of the child being stillborn from the inevitable compression of the cord while the head is passing through the lower part of the genital canal, and the fate of the child depends upon this compression lasting as short a time as possible. If the accoucheur tries to pull the head out too soon, that is, before the cervix is fully dilated, he will be unable to get it out quickly, and the child will probably die; the child may also die if he waits too long. The fate of the child, then, depends on the skill of the attendant, and in such a case there is reason why the doctor should be present while the genital canal is being dilated.

It is not necessary to say much about hæmorrhage, for every text-book faithfully impresses upon the learner the importance of early treatment, and even the most ignorant midwife knows that hæmorrhage is dangerous, and that when it occurs she ought to send for a doctor.

We have no means at present of predicting or preventing placenta prævia, nor premature detachment of the normally situated placenta, nor abnormal adhesion of placenta or chorion.

Mr. Lawson Tait proposed to treat placenta prævia by Cæsarean section, and Professor Sinclair has expressed the opinion that in this he was only

premature. I think otherwise. The chief symptom of placenta prævia is hæmorrhage, which commonly begins about the seventh month of pregnancy. If Cæsarean section is done at this time, the patient has not the prospect of a large and strong child, which is one of the chief advantages of that operation. If placenta prævia is treated early (before she has lost much blood) and rightly, the mortality attending delivery by the natural road is small. The conditions which make the mortality of placenta prævia high would make also that of Cæsarean section great.

It has been proposed to treat accidental hæmorrhage by Cæsarean section. This is sound in theory but, I fear, difficult in application. Slight cases of accidental hæmorrhage need not Cæsarean section, and in grave cases it will be difficult to arrange to do the operation with sufficient promptitude.

Craniotomy cannot be altogether banished. Take such a case as this, which I quote from a recent excellent German treatise on obstetrical operations:—You are sent for from a distance; you find a primipara at full term; she has been in labour two days; her temperature is 102°, pulse 130; the head is presenting, the foetal heart audible, the os uteri fully dilated; membranes ruptured many hours ago; the head with its greatest circumference high above the brim; the diagonal conjugate three and a half inches; the uterus is closely moulded to the shape of the child. Here, from the mother's condition, it is clear that speedy delivery is needed. As two days' labour has failed to get the head into the brim, although it has dilated the os uteri, it is practically certain that you will not do it with forceps. If you try to turn, you will find it very difficult: you may rupture the uterus, and, if you succeed in turning, you may fail to get the child's head through, and if you do the child may die during extraction. In the patient's condition Cæsarean section will be very dangerous; the only right course is to perforate without loss of time.

But this case is a mismanaged case. If the size of the pelvis and the relative size of the foetal head had been determined before labour had begun, Cæsarean section might have been done with safety.

Until our knowledge is greater than it now is, there will be an unavoidable mortality attending

childbirth. We know not yet the cause of eclampsia, and therefore we cannot prevent it. We have no treatment for it from which we can surely promise cure. While our knowledge remains what it is at present, a certain number of patients, probably about 1 in 3000, will die from eclampsia.

We know absolutely nothing about the cause of rupture of the uterus in easy labour. We cannot foretell it, nor can we prevent it. It is very rare; I know of no data from which to estimate its frequency. It ought not to be always fatal; but it will cause some deaths.

These are the ways in which, I think, the practice of midwifery ought, guided by modern knowledge, to be changed:—Recognition of the fact that a medical man's best service to a parturient woman is to early foresee difficulty in delivery and prevent it, and that this is much better than treating it; recognition, also, of the fact that if mechanical difficulty is known to be absent, and the patient is in good health, parturition is so certain to be safely completed that there is no need for the labour to be watched throughout by a highly educated man: a woman can easily be taught to do all that is required in such a case. That there is no inherent and necessary mortality in Cæsarean section; and that, in the future, it is probable that in cases in which mechanical conditions are known to be present which will prevent the safe delivery by the natural route of a full-sized child, Cæsarean section before the onset of labour will be the chosen method of delivery.

Diseases of the Tongue. (By Mr. H. T. Butlin and Mr. W. G. Spencer.)—A new and enlarged edition of this book has been issued by Cassell and Co. The present work forms a more complete treatise on the diseases of the tongue than the clinical manual which in the first instance Mr. Butlin prepared. The volume is well illustrated by eight chromo-lithographs and thirty-six engravings. To illustrate how a practitioner meeting for the first time one of the rarer affections of the tongue would know where and under what circumstances similar cases have been seen before, the following passage is taken:

"*The hairy black tongue (nigrities-hyperkeratosis linguae).*—This is a condition which has excited a great deal of interest, although its clinical import-

ance is very slight. It is produced by an overgrowth of the epidermis of the filiform papillæ, but there is no production of true hairs. The colour is often black, but it may be a sepia brown or yellow (Dinkler) or even blue (Mourek). The colour has nothing to do with the overgrowth of the papillæ, but is due to the organisms clinging to them. The black patch is almost always noticed in the middle of the dorsum of the tongue, usually immediately in front of the V formed by the circumvallate papillæ; it is darkest at the centre, and fades towards the edge of the patch, where it becomes a light brown. In exceptional cases the situation is different. In Lediard's case (the specimen is in the Royal College of Surgeons' Museum, 2266A) the patch was near the end of the tongue; in the case described by Curtis the patch had been noticed a week, and was situated behind the circumvallate papillæ. The discoloured area is at first of small size, but extends, as a rule, very slowly, until it covers a large portion of the dorsum of the tongue, lasting two or more weeks up to as many months. The affection disappears little by little from the circumference towards the centre, the colour fading to a brownish-yellow at the borders of the area, and the excess of epithelium separates by desquamation. The same series of phenomena may be repeated. In some cases only the discoloration has been noticed, but generally the papillæ are enlarged and elongated, although it must not be forgotten that very large and long papillæ are the rule in the tongues of some persons as great smoothness is in the tongues of others. The same wide differences are noticed in the tongues of animals; Raynaud described a case in which the surface of the area looked like a field of corn laid by the wind and rain. Sendziak saw a man in whom the papillæ appeared like bristly hairs, which he was accustomed to shave off, but they quickly grew again. He found that they consisted of very long filiform papillæ, composed of epithelial scales, in appearance like the stem of a plant covered with bracts."

The authors have done well to continue the encyclopædic method in the arrangement of this book; this disposition is handy, and contributes in no small measure towards the convenience of the reader. Among the many additions deserving of special notice, space only allows us to draw attention to the chapter on the anatomy of the tongue.

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A CLINICAL LECTURE

ON

PULMONARY EXCAVATION.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, December 18th, 1901.

By S. H. HABERSHON, M.D., F.R.C.P.

LADIES AND GENTLEMEN,—The subject this afternoon is pulmonary excavation. An excavation or cavity in the lung may be produced in the course of pulmonary tuberculosis in any stage of the disease. The pathological process by which necrosis or death of the tissues takes place, a process which usually results in softening and discharge of the necrotic tissue, is twofold. The toxins developed by the growth of the tubercle bacillus not only produce an irritation at the site of the focus of disease which results in local broncho-pneumonia, but a form of coagulation necrosis which converts the cell contents of the alveoli into what we usually know as caseous material. The macroscopic appearance of such a necrosis is very striking. The affected area, which at first presents the appearance of an ordinary broncho-pneumonia, becomes yellow and solid, and exhibits much of the character and colour of cheese. This process of caseation sooner or later terminates in softening and discharge of the lung contents through the neighbouring bronchial tubes. When once such an excavation has been formed the process is accelerated by the introduction and the growth of pyogenic micro-organisms. These organisms are the cause of active ulceration and destruction of the tissues forming the walls of cavities. Thus two pathological processes accelerate the destruction of lung tissue—the coagulation necrosis due to the tubercular toxin and the further progressive ulceration caused by foreign micro-organisms which develop in the necrotic areas. These pyogenic agents are not present in every case, but the two processes may coincide, or caseous necrosis may be the initial cause of the

excavation which is subsequently extended by ulceration. In the more chronic cases the cutting off of the blood-supply by the occlusion of the vessels no doubt accelerates the necrosis.

In a lecture delivered at this hospital, Sir Richard Douglas Powell has shown that an abscess cavity may be produced by one of these processes in an early stage of the disease, and that an acute stage, accompanied by the discharge of pus and pulmonary tissue, may be followed by quiescence, such as is exhibited when an ordinary abscess cavity discharges externally and resolves.

I may be allowed to make a few remarks on the pathology of excavation, or rather upon the gross pathology. You will see various forms of excavation here in these specimens, and they are closely connected with the physical signs which I shall hope to demonstrate to you later. Here is an example of a very acute necrosis of the lungs. There is evidence of an active sloughing process which has taken place. There is no great thickening of the pleura, the walls of the cavity are thin, and you can see at once that it is not an ordinary caseous necrosis. It has begun by caseation, but it has extended rapidly by an acute sloughing process. You see the vessels and bronchi are exposed, and lie across the cavity.

Here is another specimen in which the process was very chronic. We have a number of examples of this in the museum. You will understand that a certain time must elapse for the thickening of the pleura to take place, and one of the earliest pathological conditions in pulmonary tuberculosis is the adherence of the layers of the pleura. This, as a rule, is a protective process. If it were not so we should get pneumothorax in almost every case of tubercular infection. But in many cases gelatinous lymph is thrown out between the pleural layers, and this lymph after a time organises, and forms a solid and thick layer. This thickening of the pleura over a cavity is a condition which materially modifies some of the physical signs of the disease. Here is a specimen, of which we have many in the hospital museum, of total excavation of the lungs. Probably, for such a process to take place, there has been a sloughing acute necrosis extending beyond the caseous condition. Then we come to rather more chronic cases. I have selected these two specimens as illustrations. The first one shows moderate thickening

of the pleura. Here is a beautiful specimen of a cavity at the apex of the lung. Another very good one exhibits a very large cavity in the upper lobe, and another in the lower lobe behind. The section has evidently been made from front to back. There is an enormous thickening of the pleura, with some glands intervening. Another specimen is that upon which I made a post-mortem some years ago, which shows that when a cavity is very chronic it tends to contract. In the most extreme cases of displacement of heart the apex may be felt even at the angle of the scapula behind; I have not seen a case where the heart was in the right angle of the scapula, but I have seen the heart beating in the back at the angle of the left scapula, and one in which it was beating in the right axilla. In such case it has almost certainly been due to the contraction of a cavity. I do not think a cirrhotic lung ever produces such a profound contraction as that. In this specimen the whole of the lung is small and contracted, and an extreme fibrosis has been produced. The rest of the lung is shrunken, and consists of dense white fibrous tissue, with little areas of pigmentation. Sometimes an abscess cavity will go on ulcerating, and you have actually a sinus formed, which may penetrate through bone or through intercostal spaces. I have known a case in which a cavity perforated through the intercostal space, and the pus in it burrowed slightly underneath the skin, so that there was a flap of skin overlying the cavity, and with every cough the skin over the cavity would be bulged and blown out. This case occurred in Dr. Acland's practice. There is a cavity in part of the lung. Ulceration of its external wall has taken place through pleura and rib. Pus has evidently burrowed between the pleural layers, and this has resulted in the formation of a small pneumothorax over the cavity. The skin has been turned back, and you will observe the large cavity beneath.

Both from the clinical and pathological point of view, cavities occur in three types of cases.

1. The acute cases, in which an abscess cavity is rapidly produced, which discharges itself by the expectoration of pus or muco-pus, and eventually resolves and becomes cicatrised, and is followed by subacute or chronic symptoms.

2. The acute form of necrosis of lung in which the resisting power of the patient is feeble, or the

toxic effects of the tubercular bacillus are virulent, and the acute necrosis rapidly extends. These cases are described under the name of "phthisis gallopante," or the acute necrosis of lungs. In some cases pyogenic organisms are present and accelerate the process.

3. The third and common class is one in which the excavation proceeds gradually and occurs in the later stages of disease.

It is almost impossible to demonstrate the first two classes of excavation at a clinical lecture; the patients are acutely ill, and would suffer from excessive examination. Moreover, it is not necessary to bring such cases to the lecture room, because we are dealing with the signs of excavation, and these can be illustrated equally well in chronic cases.

I have very few words to say as to symptoms. Excessive expectoration is usually present, and in this respect we cannot regard the symptom as pathognomonic of a cavity, because it may equally occur when there are small cavities. On the other hand, in the case of quiescent cavities you will get very little expectoration. A patient who is the subject of pulmonary tuberculosis will often expectorate largely, but unless the large expectoration comes up suddenly we cannot attribute it to excavation. A dilated bronchus is far more prone to produce sudden and large expectoration, especially if the bronchiectatic cavity is sacculated. Still, in some cavities the discharge is profuse and intermittent. Hæmorrhage from the lung, unless in early stages, is usually a sign that excavation is present. A small cavity with aneurysm of a pulmonary vessel may result in serious hæmorrhage, but it is a fact that many chronic cavities produce intermittent hæmoptysis of varying severity.

Now I come to the immediate object of this demonstration—the physical signs of excavation. These are of great import, because they afford considerable aid to the prognosis of the disease. The most important sign that helps our diagnosis is that of contraction and retraction of lung. If no contraction is present we look to the symptoms to aid us in our diagnosis and prognosis. If in the early stages of the disease a cavity is rapidly formed, with acute symptoms, followed by resolution and quiescence, the prognosis is favourable. If during this early stage the symptoms remain acute, it is natural we should take an unfavourable

view of the case. In the majority of cases the prognosis is greatly aided by the amount of contraction, or, in other words, the tendency towards resolution and repair. Contraction is evidence of the increased resistance of the patient to the disease. The tubercular process instead of occurring in a person of weak resistance, and by its cavity producing proliferation of cells, which are of the embryonic type, and easily break down, occurs in a person of strong resisting power, and the cell tissue which is formed becomes organised into tissues of a definite form. This is generally fibroid tissue, and contraction is a great indication of the resistance of the patient to disease, and we look upon the process as favourable.

In the examination of a cavity I shall take the physical signs in regular order. First of all, with regard to *inspection*. The affected side of the chest is, as a rule, immobile, or there is at least an impairment of movement. There is usually flattening or depression in the subclavicular region if the disease is at all chronic. On rare occasions when a sinus is formed the skin may be raised with each inspiration. The heart's impulse may be observed out of place.

Then *palpation* aids us to some extent in determining whether the pleura over a cavity is thickened or not. Vocal vibrations may be exaggerated when the cavity is thin-walled, but if the pleura is greatly thickened it damps vocal vibrations, just as fluid does; therefore, if we find, on listening over the area, well-marked auscultatory signs associated with great impairment of resonance and diminished vocal vibrations, we may conclude that the pleura over such a cavity is very thick. Occasionally creaky or croaky râles can be felt by the hand through the chest-wall. Never forget to find out most carefully the position of the heart, because that is one of the signs which enables us to detect contraction. Remember that not only does the lung contract, but that it becomes retracted. Retraction and contraction are not the same thing. If we find the heart in almost the normal position, that is to say, the apex-beat in the fifth interspace, and perhaps a little outside the nipple line, and its dulness extending upwards to the third rib, or even higher, and no great or marked signs of dilatation of the heart, and no cyanosis of the patient, we conclude that the lung has been retracted and drawn off the

heart, leaving the heart in very much its normal position. But when the apex is moved, and the whole cardiac area is shifted upwards or to the right, then we know that contraction has also taken place. Extreme displacements of the heart in tubercular cases are almost invariably due to contraction following excavation.

With regard to *percussion*, the dulness depends on the thickness of the walls, which surrounding consolidated areas is usually great. Some teachers, such as Dr. Gee, at St. Bartholomew's, say that you never get resonance, or that you rarely get resonance, over a cavity. Broadly speaking, that is so, but there are varying degrees of dulness. The absolutely dull lung, as a rule, means that there is a cavity with dense, thickened, and fibroid walls or pleura over it. When the wall is thin there may be tympanitic, skodaic or "boxy" resonance. "Boxy" is a very convenient term to apply to skodaic resonance, because it conveys an idea which is intelligible to all; it is the same sound as is produced by percussing a hollow thing such as a box. A thin-walled cavity will sometimes produce the cracked-jar or cracked-pot sound, which is produced by the patient opening the mouth widely while the chest is percussed. When you have a cavity to deal with, the percussion note alters upon opening the mouth. The cracked-pot sound is not pathognomonic of a cavity, because it may be produced in the healthy chest of an infant, as also in other conditions.

It is rare for complete resonance to be present over a cavity, but it is most commonly produced when the excavation is deep-seated and covered by emphysematous lung. The cardiac dulness sometimes blends so entirely with the dulness of the cavity that it is very difficult by ordinary means to tell where one ends and the other begins. In this case auscultatory percussion will help to define the areas of pulmonary and cardiac dulness. I use auscultatory percussion by the finger or by a tuning-fork. The principle of auscultatory percussion is that when listening to the chest-wall with a stethoscope, and setting up vibrations, either with the tap of the finger or with a coin or tuning fork, those vibrations are not only conducted through the surface of the chest, but through the underlying media. It is a well-known principle of acoustics that if you have vibrations passing through one medium, and another medium intercepts them,

especially if the fibres of the intervening medium are in a different direction, they will considerably damp the vibrations; and if you listen beyond the intervening line you will find that they are duller and less distinctly heard. That is the principle of auscultatory percussion in the chest, and it helps us greatly, in these cases of excavation, to tell where the lobes of the lungs are, and where the heart is. You will find it difficult in some cases to determine the exact outline of the heart by auscultatory percussion. In some you must remember you have superficial cardiac dulness to deal with, also the deep cardiac dulness and the pericardium. So sometimes you may make out two or three lines where the vibrations alter. I hope to demonstrate in one or two of the cases how you can map out the lobes of the lungs, and find out where they have become altered in position or are contracted. Contraction of lung is shown by the movement upwards or to the right or left of the entire area of cardiac dulness.

When the lung is contracted the cardiac apex is usually displaced. Auscultation shows the size of the "displaced" cavity by the character of the breathing; it is the *auscultation of respiration*. The breathing is no longer bronchial only; it is bronchial with some superadded signs. The pitch may be the same, but it may exhibit an amplitude of wave length imparting a character which is usually described by the term "hollow." You know that you may have two notes of the same pitch, but the columns of air producing the note may be very different in size. There are various types of breathing in cavitation. All gradations occur from bronchial to amphoric breathing, and the terms I usually employ to describe the various types of breathing are "broncho-cavernous," "cavernous," "tubulo-cavernous," "tubulo-amphoric," and "amphoric" breathing. All these terms depend on the pitch and amplitude of the sounds. The greatest degree is known as amphoric breathing, and that is like the sound produced by blowing into an amphora or large jar. You only find that in the cases of very large cavities, and in those cases the patient is acutely ill. There are some such in the hospital, but I cannot have them down; excessive examination is very trying to them, so you will seldom be able to hear these amphoric sounds except at the bedside.

Another kind of breathing is occasionally caused

by the entrance of air being partially obstructed. It has been known by the name of *metamorphosing breathing*, and it implies that the air entering the cavity will produce a harsh loud sound at the beginning of respiration, altering to a softer breathing in the middle of the respiratory effort. I do not know if we can conjecture what the cause is. It may be that owing to contraction there is a small opening into a cavity, which becomes partly plugged with mucus, and that that plug gives way.

There is another curious character of breathing which is occasionally heard. It is heard more often in cases of bronchiectasis, and you know that cases of bronchiectasis often simulate cases with a cavity. It is called the "*veiled puff*" of Laennec. After having once heard it you will never forget it. The breathing begins moderately softly, and then you hear a sudden puff following the cavernous breathing. I have heard it several times in bronchiectasis, and it is regarded by some as pathognomonic of bronchiectasis, but in a case we had the other day it was present to a modified extent in an ordinary cavity. It is sometimes heard in cases of pulmonary excavation. It consists of a harsh inspiration suddenly yielding in its course to a short and sharp puff. It does not differ much from the character of breathing just described as metamorphosing, but the sudden change occurs at the end of inspiration. One of the cases I show you presents this form of respiratory murmur.

Next, I want to speak of the *auscultation of the cough*. There are two characteristic conditions that I regard as pathognomonic of a cavity; we teach them in this hospital, but you do not hear much of them elsewhere, namely "*post-tussic suction*," and "*post-tussic râles*."

Post-tussic suction is presented to a marked extent in several of the cases shown to you. After the cough, which is a violent expiratory effort, and before the next inspiration, air rushes into the cavity with a sucking sound, similar to that produced when air is drawn through the lips, partially closed. The cavity is suddenly compressed by the cough and forced expiration. Its walls are elastic, though somewhat rigid. They are suddenly compressed and expel the air contained in the cavity, but they suddenly expand again, by virtue of their elasticity, and create a vacuum, which is only filled by the sudden inrush of air. This produces the whiffing sound of entrance of air. Occasionally,

and not infrequently, this inrush of air only produces a rattling or disturbance of the moist contents of the cavity, and to this I apply the term "*post-tussic râles*." Both these physical signs occur only in the presence of an excavation, and I regard them as the only pathognomonic indications of a cavity.

Next we come to the adventitious sounds. There are râles of all kinds. When the physical signs of a cavity are not very marked, it is very important to notice the size of the crepitant sounds or râles. They are almost always of the gurgling type or of the large type. Large crepitant râles or gurgling râles are a sign of cavity, though not always a cavity in the lung; they may be due to a dilated bronchial tube. You may also find large creaking sounds, there may even be croaking sounds, such as are present in bronchiectasis. Occasionally these are synchronous with the heart-beats, and I have heard a clicking sound, which is produced by the impulse conveyed to the cavity by the cardiac impulse, and closely resembles the ticking of a clock. I remember hearing in a huge cavity a tick as if there were a clock in the man's chest. It is easy to understand that the dragging of the heart will disturb either some adhesion or some mucus. Râles synchronous with the heart-beat are not uncommon when the cavity is large and the pleural and pericardial layers are adherent. The heart-sounds are frequently heard with exaggerated intensity over a cavity.

Sometimes it is very difficult to distinguish a large cavity from a pneumothorax, for the reason that a bell-sound (produced by auscultatory percussion) is often present, and metallic tinkles, metallic echo, and amphoric breathing may be present. These signs are common to a large cavity and to a pneumothorax. If fluid is also present it aids the diagnosis materially, because when air is present in the pleural cavity, and also fluid, the mobility of the fluid, which shifts with the movement of the body, is an unerring sign of pneumothorax. In the absence of this aid to diagnosis it is sometimes a matter of extreme difficulty to distinguish between a large cavity and a pneumothorax, especially if there is no displacement of heart.

Lastly, the auscultation of the voice-sounds. Vocal resonance is increased, and we usually have the exaggerated voice resonance, known by the

name of *pectoriloquy*. This is better heard when a whisper is used to elicit the sound.

Now I have come to the end of the physical signs; those which I have described all put together will easily lead to the diagnosis of a pulmonary cavity in tuberculosis. There are some cases in which the cavities are very small and far below the surface. These cannot be detected at all. Especially is that the case in an emphysematous lung, where the emphysema covers over the cavity. I have performed a post-mortem upon a case in this hospital in which the patient was known to have pulmonary tuberculosis, owing to the presence of tubercle bacilli. On the diagrams the whole chest was described as resonant or fairly so, and nothing but bronchitic signs were present. On the post-mortem table the lung was riddled throughout with cavities, some of which were almost as large as a walnut, but they were deep in the lung, and over them was emphysematous lung tissue. No evidence could be obtained of their presence by any of the diagnostic signs. There is only one sign which will help us where the cavity shows no other marks of its presence, and that is the physical sign of post-tussic suction. I am a great believer in the importance of that in a case of cavity, and I must say that wherever I have been able to test it we have always found a cavity.

I regret that I cannot demonstrate to-day any case of a cavity producing metallic signs, but I bring you a case of pneumothorax which presents a well-marked bell-sound. This is often heard in the case of a large cavity. The patient came to us in the out-patient room, and we examined her very carefully, and concluded she had some obstruction to her right bronchus. There was a history of gradually increasing shortness of breath extending over some considerable time, and with slight impairment of resonance at the right apex. There was practically no air entry, or at all events very little; there was excessively feeble breathing, and I think the vocal vibrations were somewhat diminished. All over the right side was this impairment of resonance. We examined her face as to whether her nose indicated anything specific. I concluded there was something obstructing the left bronchus, and I think we were fairly justified in coming to that conclusion of obstruction. We suggested that there was probably some stricture of her left

bronchus, but we took her to the Röntgen ray room and could find no aneurysm or any shadow which showed pressure upon the bronchus. Dr. Acland came into the out-patient room, and at my request he examined her carefully. I suggested there was pressure on the bronchus, or some stricture, and he agreed. She was taken into the hospital, but she had not been in long before he discovered a well-marked bell-sound and a succussion splash. Therefore this is really an unusual form of pneumothorax. She is rather stout, and that added very much to the difficulty of its recognition. I have just tested her, and find the bell sound very marked.

The cases I now show you exhibit the varying degrees of cavernous breathing and pectoriloquy, and in several cases the post-tussic suction and râles are very striking. In some cases the vocal vibrations and resonance are materially diminished, and these indicate a thickening of the pleural layer. Several cases of extreme contraction of lung are shown. You will note the displacement of heart and the pseudo-hypertrophy of the opposing lung.

It is told of a Western surgeon, who had been brushing up in the clinics of New York, and was returning to his home, that he met a fellow-traveller, a native of the metropolis, who asked what had most deeply impressed him in the gynæcological work which he had seen, and that he replied, "I would rather be a snowball in hell than an ovary in New York." There is no pathologist with any degree of experience who does not realise the truth of this more forcible than elegant expression, for it is doubtful whether there is any who has not received for examination female uteri or appendages which were normal, or showed only some insignificant lesion. Not infrequently has this occurred even after the operator has received from the pathologist a report which, if he understood its meaning, should have contra-indicated operation. This is the most charitable construction which can be placed upon the removal of uteri and ovaries for insufficient reasons.—*Amer. Journ. Obstet.*, February, 1902.

THREE CLINICAL LECTURES ON NERVOUS DISEASES IN CHILDREN.

Delivered at the Medical Graduates' College and Poly-clinic.

By JAMES TAYLOR, M.A., M.D., F.R.C.P.,

Physician to the Hospital for the Paralysed and Epileptic, Queen Square; Physician to the Royal Ophthalmic Hospital, Moorfields; and to the North-Eastern Hospital for Children.

LECTURE II.

GENTLEMEN,—To-day I propose to resume the subject of my lecture of last week, and I wish first to say a few words on the paralysis which results from intra-cranial tumours in children, and from intra-cranial tumours in young adults. It is hardly necessary to say that naturally all the symptoms resulting from the presence of a tumour in a child or in a young adult depend entirely upon the position of the tumour and the character of the tumour. Still, there are certain particular features, I think, which are peculiar in children in reference to the symptomatology of intra-cranial tumours.

In the first place I think that infiltrating tumours in the neighbourhood of the pons are unusually common in young children; I mean in children below the age of ten. As you know, the condition used to be described as hypertrophy of the pons, because, when seen post mortem, the appearance closely resembled simple enlargement of that part of the brain. As regards the symptomatology of tumours generally, in children I should say that they are probably distinguished from the symptoms in adults by a less degree of severity; for example, in a child you very rarely get the intensely severe headache and the very severe vomiting which are so common in the adult, and I think the reason for that is probably because of the much greater power of yielding that the skull of the infant or young adult possesses.

You know the sutures in the skull of a child are not nearly so closely united as in that of the adult, and consequently they are able to separate, and in many cases, especially in children of three to four years of age, we see a tremendous expansion of the head taking place as the result of intra-cranial pressure, and we see the sutures gape and a rudi-

mentary parchment-like bone formed between the sutures. I have noticed that appearance in a good many cases of children who have died from cerebral tumours, and I think it is entirely due to the intra-cranial pressure being so severe and causing the sutures to gape, and I think it also probably accounts for the less severe headache that you find in children, and also for the slighter severity of the vomiting. I might just say a word about cerebellar tumours in children. Of course they occur in children as well as in adults. According to their position in the cerebellum they give rise to different symptoms. If a tumour is entirely restricted to one of the lateral lobes of the cerebellum there may be no local symptoms, no staggering, only headache, sickness, and optic neuritis—an optic neuritis which very often goes on to complete atrophy and consequent blindness. If, on the other hand, you have a tumour which encroaches upon the middle lobe you will probably have the rolling cerebellar gait which is supposed to be characteristic of a cerebellar tumour, yet, as I hope to show you next week from a post-mortem specimen, you may have a tumour which is, so far as one can judge, almost completely restricted to the lateral lobe, which must still have caused considerable pressure on the middle lobe, in which there were no symptoms at all of any pressure on the middle lobe, but only the general symptoms characterising intra-cranial tumour; that is to say, headache, sickness, and optic neuritis. One point which I would like to mention with reference to cerebellar tumours in children, which is true also of most cases of cerebellar tumour in adults, and that is that the tumours in that region are very often, I might say almost invariably, associated with a very intense form of optic neuritis—a greatly swollen disc, numerous hæmorrhages, and also that curious arrangement of white streaks radiating from the macula lutea, which is so common in albuminuric retinitis; so that in many cases of cerebellar tumour one of the first questions that one asks after an examination of the eyes is, Has this patient any albumin in the urine? because the appearance of the optic disc, and of the fundus generally, is so very closely suggestive of the retinitis which is associated with albuminuria.

I might here just refer to the disease which is known as cerebellar ataxy, which in so many par-

ticulars resembles closely the disease of which we shall speak presently, called Friedreich's ataxy. In cases of cerebellar ataxy the knee-jerks are very active, and there is unsteady rolling gait which is very like that seen in Friedreich's ataxy, and post mortem there has been found in some cases a very remarkable degeneration of the cells in the cortex cerebelli, so that presumably the disease is associated with some maldevelopment of the cerebellum or with some tendency in the cerebellar cells which leads them to die early. The disease is characterised in its symptoms by the rolling gait, by the ataxy, by the exaggeration of the reflexes, and by these well-marked signs it is distinguished from Friedreich's ataxy. Then I might also mention a suspicion that there is a class of cases very closely analogous to those of which we spoke last week, in which we have a decay of the pyramidal cells going on in a child after a period of apparently normal health; we occasionally see a child who is brought on account of the difficulty it has in walking, or perhaps a difficulty in using its hands, which are unsteady and shaky, or the child rolls about, yet the intelligence is perfectly intact, and in such cases I think there is reason to suspect that we are dealing with a cell degeneration in the cerebellum similar in character, and possibly also similar in its ætiology, to the cell degeneration which takes place in the pyramidal cells in those cases to which I made reference last week. So much for diseases which affect the intra-cranial contents.

We now pass on to consider diseases of the spinal cord occurring in children and in young adults. Of course by far the most common disease we meet with in children is the disease of the spinal cord known as infantile paralysis. It is also known as the essential paralysis of children, and it is also known as anterior poliomyelitis. I think it is very likely that it is desirable to keep to the name anterior poliomyelitis, although, as I shall have to say presently, there is reason to suspect that the disease is really not an inflammatory one, but a disease depending on vascular obstruction, but the name anterior poliomyelitis is a good one because it does not limit the occurrence of the disease to children merely, and we now know that the disease does affect adults, and it is as well, perhaps, to have a general name like anterior poliomyelitis to indicate this

important point. I have notes of 113 cases which I have analysed, and possibly some of the points which came out in the analysis may be interesting while we are considering this disease.

First of all, in the 113 cases in whom I could accurately ascertain the ages of the onset of the paralysis, in 105 cases it occurred in the first ten years of life, in 4 it occurred in the second ten years of life, in 1 it occurred in the third ten years of life, in 2 in the fourth ten years of life, and in 1 in the sixth ten years of life. So that we have the disease occurring as late as the sixtieth year. Taking, then, those occurring in the first ten years of life, which were 105, of these 33 occurred in the first year, 37 in the second year, 14 in the third year, 8 in the fourth year, 6 in the fifth year, 4 in the sixth year, 2 in the seventh year, and 1 in the ninth year. So that we may say from the second year onwards there seems to be a greater tendency towards the restriction of the occurrence of this disease. In the first three years, out of 105 cases, as many as 84 occurred, leaving a very small number (21 cases) to occur in the years from the fourth to the ninth. This disease may naturally enough be described as the essential paralysis of children, or as infantile paralysis, and there is no doubt that the great majority of cases occur in the first three years of life, and of course the preponderating number occurs in the first ten years of life. Now as regards the mode of onset of the disease; first of all, I would say that there are two or three different modes of its occurrence. The most general is probably that in which the child is ill; there is a general feeling of malaise and a high temperature, and there is sometimes the occurrence of convulsions. I think, however, that convulsions are rare; at all events, in only five of this large series of cases did it occur, so that it did not occur, at all events, oftener than about once in every twenty cases. To sum up the symptoms briefly, you have the general malaise, the high temperature, and you often have also a certain amount of pain, and this is rather important, because infantile paralysis is sometimes said to occur after rheumatic fever; but in some cases, at all events, in which I was able to make observation, in which such a cause has been alluded to, the paralysis was only discovered when the child was allowed to get up after being ill, and it seemed to me that there was strong reason to

suspect that the disease which actually ushered in the paralysis was nothing more than the acute onset of the paralysis itself, and that the pains which were supposed to be rheumatic were really of the nature of neuritic pains, which are not at all uncommon at the onset of the trouble. Besides this mode of acute onset, which is the most frequent mode of onset in children, you may also have the onset occurring in the night in a child who the evening before was perfectly well, and who had previously had no constitutional disturbance, but in the morning was found to be paralysed in one or both limbs; usually the paralysis in such a case is restricted to one limb. You may also have the occurrence of a slight accident or injury as precursor of the paralysis. In several cases we have the history given us that the child has had a fall, when its ankle was twisted, but it has otherwise been apparently all right, and has been able to walk about, but on the following day is found to be paralysed, and when the case comes under observation the paralysis is of the nature we are now considering. Another class of cases is characterised by its onset occurring after the child has been subject to unusual exertion; for example, one child whom I saw, and whose case is included in the series I have given, was a little girl *æt.* 5, who had been taken for a long walk of over eight miles by her father one summer afternoon, and the next day she became totally paralysed in both her legs. These four modes of onset, I think, embrace most of the cases of this disease. To recapitulate, there is—(1) onset with constitutional disturbance, malaise, and high temperature; (2) in the night without any premonitory symptoms or any constitutional disturbance; (3) after some slight accident or injury, to which no importance is attached at the time, but which nevertheless probably has some bearing on the onset of the disease; and (4) after undue physical exertion.

I have spoken about the pains which attend the onset of the disease in certain cases, and this is important, because these pains are completely absent in some cases, but occasionally they are a very marked feature in those in which they are present. A few years ago it was pointed out by Sir William Gowers, I think in a paper read at the Clinical Society, that in all probability the symptoms in those cases indicated not only that there is some involvement of the anterior horns at

that time, but that there is also some neuritis, some inflammation of the peripheral nerves attending the onset of the paralysis, and that this neuritis really accounts for the pains which are present. I shall have more to say about that presently with reference to the *ætiology* of the disease, but I may say in passing that there is reason to suspect that we have also most likely a class of cases similar in most respects to cases of infantile paralysis, in which the inflammation is limited to the nerves, and those are cases which completely recover.

As regards the time of onset of the disease, that is very important, because infantile paralysis is essentially a disease of the hot weather; it is a disease which rarely occurs during the cold weather, and in sixty-nine of my cases in which I was able to accurately ascertain the month of the year in which the onset occurred, this fact comes out very strongly. Two cases occurred in January; 4 in February; none in March or April; 3 in May; 7 in June; 16 in July; 17 in August; 6 in September; 6 in October; 5 in November; and 3 in December. No less than 40 out of the 69 (nearly 58 per cent.) occurred in June, July, and August, so that there must be some proclivity, at all events, to put it not too strongly, to the occurrence of the disease in these months. It not at all uncommonly occurs in a child who has been paddling in the sea. However, the point I was going to make just now was the occurrence of this particular trouble in association with the onset, or at all events with the presence, of a somewhat high temperature. It may be interesting to add that exactly the same incidence of the disease comes out in other countries, in America for example.

Now as to the parts affected by the paralysis. One general statement I may make, and that is that the extent of the paralysis is much greater at the onset of the disease than it ultimately becomes in the great majority of cases, that is to say, where in some cases both legs are at first paralysed, the paralysis ultimately becomes restricted to only one leg, and with reference to parts ultimately affected, I have notes of 115 cases, and in this series one leg was affected in 52 patients. Curiously enough the right leg was affected in nearly as many cases as the left, the figures being left 24, right 18. In 47 both legs were affected, in 15 the arms only were affected, the left being paralysed in 8,

and the right in 7 cases ; both arms were affected in 3 ; in 2 cases one arm and one leg were affected ; in one of these cases, one arm was affected on one side and the leg on the other, and in the other case the disease was hemiplegic in character, that is to say, it affected the arm and leg on the same side, and that is important, or rather I should say the infrequency of such a case is important, because the distinction between infantile paralysis and infantile hemiplegia scarcely presents any difficulty ; first of all because in the former the hemiplegic form very rarely occurs, and secondly because, as we shall see, the state of the limbs is totally different in the two diseases.

Now as to the state of the affected parts when the patient comes under observation. First of all there is the wasting noticeable ; the limb—or limbs as the case may be—is wasted, and there is also loss of reflex in the muscles which are affected. It is very important to remember this limitation, and that you do not get naturally in infantile paralysis as the result of the disease loss of the knee-jerk in an affected limb, you only get loss of the knee-jerk if the vastus internus is one of the muscles involved. I have seen numerous cases of infantile paralysis in which the paralysis was limited to the muscles below the knee in which the knee-jerk was quite active. It has been said that infantile paralysis is one of those diseases which, when it affects the legs, produces absence of knee-jerk, but that is not the case. The knee-jerk is absent only if the muscles which subserve the knee-jerk are involved in the paralysis. Besides wasting, it will also be noticed that there is some interference with the circulation of the limb. The limb is nearly always cold unless it is thoroughly wrapped up and kept as warm as possible, and it is nearly always cyanosed. It seems as if there were some vaso-motor paralysis present ; so that we have the wasting, and the vaso-motor disturbance resulting in cyanosis of the limb. Then, of course, we have the flaccidity which results from the absence of the muscles, or rather from the paralysis of the muscles at first, and we may also have certain deformities produced by the tendency of the muscles which escape paralysis to contract, and consequently we have some talipes very frequently occurring, either varus or equinus or valgus. If in any particular case we have paralysis of the anterior tibial muscles, they being the natural

opponents of the gastrocnemius, the latter muscle is unopposed, and by its contraction gives rise to talipes equinus.

In regard to the ætiology of the disease, it must first be stated as a well-known fact that there are frequent epidemics of this disease. Perhaps I ought not to state it in this way, but rather to say that epidemics of this disease occasionally occur, and it is not uncommon to find that the disease has affected simultaneously two members of the same family. Then it is also not unimportant to remember what I said last week that in at least one of the cases in which three members of the family were affected with paralysis, in two of the children the paralysis was of this type (anterior poliomyelitis), and in the other child the paralysis was of the hemiplegic type, that is to say, in two the spinal cord was affected and in one the cerebrum was affected. It seems to me that the occurrence of these epidemics and the occurrence simultaneously of more than one case in the same family rather suggests to us the possibility that some toxic influence is at work giving rise to the paralysis which takes place. And if we assume or suppose that such a poison is present at any time we can, I think, understand some of the variations in the symptomatology which occur in connection with the onset of the disease. We can easily, for example, understand the onset of the trouble associated with constitutional symptoms ; we can also, I think, understand the association with the symptoms of paralysis, of pain if we have the same toxic agent acting upon the peripheral nerves which acts upon the anterior horns, and we can also understand how it is that occasionally the influence of such a toxin is limited to its influence upon the peripheral nerves. Taking all these points into consideration, it seems to me that in this disease we are probably dealing with some poison, very likely some chemical poison, although we cannot neglect the effect of microbes in the causation of this chemical poison ; it may, for example, be similar to the poison which follows diphtheria and which gives rise to diphtheritic paralysis. But that, of course, is merely a surmise ; at all events, it seems to me that the occurrence of this disease in epidemic form, and its occurrence simultaneously in several members of the same family, rather point to the likelihood of infantile paralysis being a toxic disease.

Now as to the actual morbid anatomy or pathology which we find in those cases that have been examined. Of course, the condition will vary with the date after the onset of the paralysis at which the case has died. If, for example, we examine an old case of infantile paralysis, that is to say, a case in which the disease has existed for a long time, naturally we shall find that the spinal cord is wasted; we find that the anterior horn-cells have disappeared to a very large extent, and we find that the whole cord on the affected side, if only one side is affected, is shrunken. If, on the other hand, we have a case which dies at or soon after the onset of the attack—and, of course, some cases, on account of the severity of the attack, do die shortly after the onset,—we find that there is usually some blocking of the vessels, that the anterior spinal arteries which bifurcate in the anterior fissure to supply each anterior horn are thrombosed, and that as a result of this thrombosis, the anterior horn-cells are disintegrated and disorganised, and the spinal cord itself is generally inflamed; and I think that possibly the œdema or the inflammation, whichever it is, which surrounds the actual seat of the lesion in such a case probably accounts for the fact already mentioned, namely, that the paralysis is often much more severe and extensive at the onset than it is ultimately; such œdema being sufficient to cause interference with the function of certain structures, but when the œdema has disappeared these structures resume their functions if they have not been actually involved in the lesion, that is to say, if they do not actually come within the area of blood-supply of the vessel which is blocked. Such a view would probably account for what is said to be present in some cases, a certain degree of impairment of sensibility of the affected limb in the early stages, for there is occasionally a history that on the onset of the paralysis there was present a certain degree of interference with sensation, an impairment which has passed away before the patient is seen. Such, then, are the most recent views as to the morbid anatomy, namely, that while the anterior horn-cells are disorganised the disorganisation really depends upon a blocking of the vessels, and that the extent of the paralysis depends upon the degree of blocking which has taken place, and whether it occurs on one or both sides depends upon whether the branches which

go to supply each anterior horn are involved or only one branch.

Now as regards diagnosis. The diagnosis of these cases has to be made from two other conditions as a rule. First of all, from cerebral palsy, and in the second place, from myopathy. From cerebral palsy it seems to me that the distinction is quite easy; it (the spinal disease) is very rarely hemiplegic, and in only one out of 115 cases was there any possibility of mistaking the case for one of ordinary cerebral hemiplegia, because in only one of those cases were the arm and leg affected on the same side; but even if there were any frequent occurrence of the hemiplegic condition, the fact that the limbs are flaccid, that the reflexes are abolished, at all events if the muscles subserving them are affected, and that the limbs are cold and cyanosed, and also the history of the onset would probably be sufficient to distinguish the two diseases. From myopathy it is occasionally more difficult, but in myopathy, again, you have the history of a gradual onset of paralysis to guide you, whereas in infantile paralysis the onset is practically always sudden, and in myopathy, too, you have a much more wide-spread condition to deal with, and one which is not restricted as infantile paralysis is to the affection of one limb or, perhaps, of two.

And now as regards the treatment. The treatment must in this disease resolve itself into two parts: first of all, that at the onset of the paralysis; and secondly, that which is to be adopted when the condition has become established, and when all that is possible has been done for restoring the function of the paralysed parts. As regards the treatment at the time of onset, that would be on general lines, and the condition would be treated as if it were a general fever. In every case that comes under your care with high temperature and malaise, especially if associated with any pain in the limbs occurring in a child, it is always well to have the possibility of the occurrence of infantile paralysis before your mind. It is a thing which is often overlooked, and which is frequently only recognised when the acute process has passed off and the child is beginning to get about again, so that, I repeat, in every case of feebleness and pain, especially when occurring in a child, it is advisable to have the possibility of the occurrence of infantile paralysis in one's mind. I do not think at this

stage of the disease that any specific or particular drug is to be recommended. Some state that belladonna is useful, but I should think that the recovery of a child who did well after taking belladonna in this stage is probably merely a coincidence, and, as a matter of fact, I should think that at the time of the onset of the disease, when the child is feverish, the less active the treatment is the better. The drug which naturally suggests itself is salicylate of soda, with a view to eliminate the poison or counteract its effect, and also with a view to relieving whatever pains are present. Then as to the treatment afterwards; this, as I have said, resolves itself into an attempt to restore the function of the paralysed parts. In the first place, massage and passive movements are extremely important in order to prevent contractures from taking place, and also to favour the nutrition of the muscles. Electricity is, of course, important, but not to the same degree that massage is, and I think that in this disease electricity, if used injudiciously, often does harm; for example, some children are very much frightened at the use of the battery and at the effect which it produces and the pain it causes, and if the child screams and makes a scene each time it has the battery it is better not to use it at all. If, however, the battery is used it ought to be used in such a way as to cause a contraction in the muscles which do not respond to voluntary efforts; if it does this, it is the best kind of exercise that the muscles can get, so that the use of the *constant* current is clearly indicated. This latter can be used without upsetting and disturbing the child too much, but in applying it it is always necessary first of all to gain the child's confidence, and if only to accustom the child to the presence of the battery, it is well to begin by putting the poles on without allowing the current to pass at all. You may even find it necessary to do this more than once, and in all cases it is necessary to do it the first time the battery is used. The strength can be gradually increased till you get, if possible, a visible contraction, but if you are sure a good current is passing it is not necessary to get a *visible* contraction. If a good current is passing and the skin is well moistened you may be quite sure that contraction is taking place in any muscular fibres which are still susceptible to galvanism. These are really the only points to be mentioned as regards the treatment in the after

condition. The treatment which is directed to favour the nutrition of the muscles which have suffered and to prevent contraction taking place is that which is necessary, and the only means at our disposal are those which I have mentioned, namely, massage, by far the most important part of the treatment; and, secondly, electricity, the use of the constant current, which has the same effect. Of course, we must also naturally look to the child's condition of general health.

I need only just refer to the chronic disease as it occurs affecting the anterior horn-cells. It is extremely rare in children, but it does sometimes occur, and a form of the disease which has been described within the last few years is that in which there has been wasting of the anterior horns, associated with a condition of the muscles which is similar to that which is found in myopathy. It seems as if this disease which I am now mentioning were a connecting link between the true spinal cord diseases and the myopathies, diseases which are supposed to have no condition of the spinal cord underlying their manifestations. I repeat that this disease to which I am referring is characterised by changes in the muscles similar to those which occur in myopathy, like pseudo-hypertrophic paralysis, and also by changes in the anterior horn-cells similar to those occurring in the progressive muscular atrophy of the adult.

Then we come next to consider the occurrence of acute myelitis, and this may be dealt with very shortly. Myelitis does occur in young girls, sometimes, but very rarely, in quite young children, although not in infants, at all events in my experience. In girls it occurs most commonly between the ages of sixteen and twenty-five, more especially in girls who are chlorotic. In such patients the condition underlying the myelitis is probably one of thrombosis. We know that in chlorotic girls thrombosis does occur in the vessels of the cerebrum, giving rise to hemiplegia. The occurrence of myelitis in girls of about the same age and under the same conditions is probably to be ascribed to thrombosis occurring in spinal veins. The onset of the paralysis is characterised by a certain febrile disturbance, and sometimes by pain in the back, and sometimes also by a gradually increasing weakness in the limbs which are paralysed, but often the disease is extremely sudden in onset, and its suddenness is often such

as to suggest hæmorrhage. Thus a girl who a couple of hours before was going about absolutely well, at the end of that time has become paralysed in all four limbs. One extremely important point is the tendency to the rapid formation of bedsores. You may have within a few hours the formation of a very large bed sore; this must be recognised, because every measure must be taken to prevent the occurrence of such trophic disturbance as soon as possible after the commencement of the illness. Even after a patient with this disease has been put upon a water-bed, and every possible precaution has been taken against the occurrence of bedsores, you must not be surprised, however much you may be disappointed, at the occurrence of a bed sore. It does not imply that the nursing is bad, but that in some cases it is impossible to prevent them, for the skin gets so tender, and the tendency to trophic disturbance becomes so great, that the occurrence of a certain amount of breaking of the skin is almost inevitable. The extent of the paralysis will entirely depend upon the position at which the lesion has taken place. Undoubtedly it is most common in the lumbar region, but it does also occur in the cervical region, and from the experience of certain cases it seems to me that there has been a simultaneous occurrence of the diseased condition we believe to be thrombosis in both cervical and lumbar regions. After a time you get descending sclerosis if you have the paralysis above the lumbar region, and in all cases the bladder and the rectum are likely to be interfered with.

Another form of myelitis in children is the pressure myelitis, occurring usually as the result of caries of the spine, and sometimes as the result of a tumour. The onset is characterised by its gradual occurrence, and you can often find no deformity in the bone of the spine to account for it, although as a rule, when pressure paralysis occurs, there is some deformity in the form of curvature of the spine to account for the pressure. As the disease is characterised by the paralysis, by the excessive reflexes, and usually by some impairment of sensibility and by interference with the sphincters and by the bony deformity, it can be easily enough distinguished from most other conditions. Of course, if you have curvature of the spine in a patient who comes before you complaining of weakness of the legs and inability to walk,

the diagnosis is easy, and such cases are not difficult to distinguish from cases to which I have just referred of ordinary myelitis in young girls, but in the latter class of cases the tendency to the formation of bedsores is extremely marked, whereas in patients with spinal caries and spinal paraplegia the tendency to bedsores is extremely slight. I should just mention the possible sudden occurrence of paralysis in cases of tumour of the cord and meninges—cases which are, of course, usually characterised by pain and by a gradual onset of paralysis—still, one must not be induced, even by the occurrence of a sudden paralysis, to ignore the possibility of a tumour of the cord as its cause. I remember a little girl in hospital, who was in a convent school, and who, while kneeling at prayers, found when she tried to get up that she could not; she was completely paralysed in both legs, and remained so, and we naturally thought that the case was one of myelitis as a result of chlorosis, because she was anæmic. But after a few months there was external evidence of a growth of the spinal column, and when she died we found a large sarcoma involving the spinal cord, which had evidently set up myelitis, the cord being very much involved in the tumour mass itself.

CLINICAL REMARKS UPON SOME CASES OF MOVABLE KIDNEY AT THE LONDON HOSPITAL.

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THIS afternoon, gentlemen, I wish to make a few remarks upon some of the cases of movable kidney of which we have recently had so many interesting examples in our wards. I mean, of course, what is ordinarily called movable kidney, not that variety which is sometimes distinguished as "floating kidney," and which is said to possess a mesonephron of its own, for this I have never seen, and I do not understand how such a structure as a mesonephron formed of peritoneum can be developed.

In the first place it is necessary to define what we mean by a movable kidney. All kidneys are movable. In a great many people the normal up-

ward and downward range of movement is more than an inch, and may be as much as two inches when the patient is standing upright and takes a deep breath. Such a kidney, if the thorax happens to be long and narrow, can often be grasped below the ribs; but even if the lower half can be grasped in this way it does not follow that the kidney is movable in the pathological sense of the term. It merely means that it is situated rather lower down than usual. The range of movement only becomes pathological when the kidney, whether it is high up or low down, fails to follow the upward movement of the diaphragm. If it descends with the diaphragm, and does not spontaneously resume its former position as soon as the diaphragm relaxes, the range is pathological, and the kidney deserves to be called movable.

But this is not the only form of movable kidney. There is another variety, no less important, though not so often recognised, in which the kidney need not descend farther than it does under perfectly normal conditions. It is only the upper end that moves. This drops forwards, undergoing a kind of anteversion, so that the long axis of the kidney becomes changed from the vertical to an almost horizontal direction, and the upper end, when the patient stands upright and the diaphragm and liver descend, is pressed against the posterior aspect of the pylorus or duodenum, or common bile-duct. Of this peculiar displacement, which may or may not be associated with pathological descent of the kidney, we have had quite recently several very interesting examples. It is not perhaps so serious, so far as the kidney itself is concerned, as the other more common form of movable kidney; that is to say, there may not be the same risk of congestion, or of hydronephrosis, from kinking of the pedicle, but, as our cases have shown, it may lead to all the symptoms of gastric ulcer, or give rise to severe attacks of biliary colic, and it is not at all improbable that it is the real, although unrecognised, cause of many of those cases of dyspepsia and distress after food which are so commonly met with and are so difficult to treat in middle-aged women.

One of our cases was a married woman *æt.* 44, who was in Royal Ward a few months ago. She had had nine children, six of whom were living. For the last twenty years she had suffered from pain in the epigastrium, shooting round to the

back and shoulders. The pain invariably came on a quarter to half an hour after meals. Solid food made it worse. Vomiting was frequent, and was rather encouraged, as it relieved the pain. Scarcely a day passed without at least one attack, and for the last nine months there had been no respite. Twenty-one months ago there had been three attacks of hæmatemesis, the amount said to have been as much as three quarts, and there was melæna at the same time. The abdomen was large and flabby. According to the patient's account she had been getting thinner. The stomach was not dilated or displaced, the lower border being situated about two inches above the umbilicus. There was a little tenderness on deep pressure to the right of the epigastrium, but no tumour could be felt. Both kidneys were movable, the right one in particular descending so far, when the patient strained or coughed, that it came quite below the thorax, and the hands could be made to meet above it. While in the ward, lying in bed waiting for operation, the vomiting, which had been more and more troublesome, and which was the immediate cause of her seeking admission, ceased entirely, and the pain after food diminished so materially that it scarcely interfered with her comfort. This led me to the conclusion that the mobility of the right kidney was the chief, if not the sole, cause of her symptoms, whether it acted mechanically, by pressing or dragging upon the duodenum and pylorus, or whether it irritated the splanchnics in some way, leading to persistent congestion of the mucous membrane of the stomach, with its attendant consequences—chronic gastritis and hæmatemesis. The right kidney accordingly was exposed through an incision in the lumbar region, all the fat around removed, and three kangaroo tendon sutures passed through it, securing it to the psoas. Iodoform gauze was packed around the outer border and lower end of the kidney to prevent any strain falling upon the sutures during vomiting, and to secure a broad surface for adhesion afterwards, and the wound was closed. The subsequent progress was perfectly satisfactory. The patient was kept in bed until the wound was sound, and then allowed to get up, wearing an abdominal belt as a precaution. There were occasional attacks of dyspepsia afterwards, but so far as the severe pain and vomiting were concerned, relief was complete. She could

perform all her ordinary household duties without inconvenience.

In two other cases that were in the same ward about the same time, the effect of the displacement of the kidney fell not so much upon the pylorus as upon the gall-bladder or the common bile-duct. One of these patients was a young woman, unmarried, *æt.* 26. The other was married, with a large family, and probably a little over forty. The history they gave was practically the same, so far as important details were concerned. In both there had been repeated attacks of colic, beginning in the right hypochondrium, and resembling mild attacks of biliary colic, but bearing little or no relation to food. They did occur after meals, it is true, but they occurred almost as frequently at other times. In the elder of the two these attacks had lasted a good deal longer and were much more severe than in the younger. In her, too, they were often attended by jaundice, which, however, rarely lasted more than two or three days. Occasionally there was sickness, but it was not sufficiently regular or frequent to enable it to be said that it was dependent upon the colic. There was tenderness in the right hypochondrium in both, especially after one of the attacks. The liver was not enlarged, and no tumour could be felt when the patient was lying down. When standing up and leaning forwards, supporting the weight of the upper part of the body upon the hands, so that the abdominal muscles were relaxed, a sensation of deep resistance was felt over the region of the gall-bladder, especially in the case of the elder patient, but the gall-bladder could not be defined. The lower end of the right kidney could easily be felt in both when they were standing upright, but it was not movable in the ordinary sense of the term; that is to say, though it descended distinctly when the patient took a deep breath, it resumed its former position as soon as the pressure of the diaphragm was taken off. But the kidney was the immediate cause of the symptoms in both, and the elder of the two gave us the clue by volunteering the statement that, if she could only get to bed and lie down for a few hours, the pain almost ceased. Very often, so she said, it returned again after she had been up a little while, especially if she had occasion to exert herself. Both patients were operated upon, if you remember,

in the same way as the one I have already mentioned to you, with complete success. The symptoms disappeared at once, and there was no further trouble.

At first sight it may strike you as curious that this peculiar displacement of the upper end of the kidney, giving rise to symptoms of such severity, should hardly ever be described in pathological records; but the same thing is true of the ordinary form of movable kidney, which has been known for years. Clinical observers described it long before it was recognised by pathologists, and the reason is the same in both cases. The kidney is not movable *post mortem*. When the body is placed upon its back the kidney drops back into its bed, and becomes fixed there by the rigor mortis, which hardens the fat and all the surrounding viscera, and securely embeds it. For the same reason the normal kidney used to be described as fixed and immovable. It is fixed and immovable after death, but it is not during life.

Another case, gentlemen, which is still in our wards, is of particular interest in connection with the question of the causation of movable kidney. An immense variety of reasons has been assigned for this, as you know, but the facts for which we have to account are clear and distinct. Movable kidney is ten times more common in the female sex than it is in the male, and ten times more common upon the right side than upon the left. In fact, it very rarely occurs upon the left side by itself, but in this our patient is an exception.

Now the forces which maintain the kidney in its place are very weak. Normally the kidney moves through a considerable range, and a slight increase in weight or a violent jerk is quite sufficient to increase this range and make it pathological. This condition of insecurity, as I have shown elsewhere, is one of the penalties attached to the erect attitude to which man has attained. In quadrupeds, in which the thorax and pelvis are narrow from side to side, and the pressure of the diaphragm comparatively slight, the kidneys are well supported by a layer of fascia which separates them from the peritoneum beneath and rests upon the viscera. In man, on the other hand, the thorax is broad from side to side, the ribs spring out laterally, the spine in the renal region is convex forwards instead of concave, the pelvis, especially in women, is enormously wide, and as a consequence the

renal recesses are open and shallow, particularly towards their lower end. In man, too, the fascia and the viscera lie in front instead of beneath, and there is nothing to hold the kidneys in place but some bands of fibrous tissue, of no great strength, which pass through their fatty envelope from their capsule to the fascia, and the general abdominal pressure. No wonder they become movable, especially in women. The wonder is that they are not always movable. There is no need to accuse the disturbance caused by pregnancy (as a matter of fact, movable kidney is quite common in young girls and even in children) or the wasting of the fatty tissue around them or a general enteroptosis affecting the intra-peritoneal viscera. These have no more to do with the development of movable kidney than the tight lacing or the wearing of high heeled boots which is one of the popular reasons assigned for it abroad. The anatomical arrangement when the body is raised into the erect position is quite sufficient.

But then why does movable kidney occur upon the right side, to the practical exclusion of the left, and why did it occur in our patient, an unmarried woman of about thirty years of age, upon the left side and not upon the right? This, as you know, is often said to be the fault of the liver, as if it were possible for the normal intra-abdominal pressure to be persistently greater in one part than in another. No, gentlemen, it is not the liver: the real cause is in the spine. It is the rotation to the left of the bodies of the lumbar vertebræ, which is the necessary result of the right dorsal convexity that nearly every one possesses. If there is a right dorsal convexity of the spine, there must be a left lumbar one to compensate for it; and if there is a left lumbar convexity, there must, from the shape of the articulations between the vertebræ, be a certain degree of rotation, so that the bodies move further from the middle line, and the spinous processes come back towards it. This, of course, brings the transverse processes on the right side forwards, while those on the left sink back; and, as a natural consequence, the shallow renal recess on the right side becomes more shallow still, while that on the left is deepened. Our patient, in whom the actual displacement of the left kidney was caused by a rather violent shock, volunteered the statement that she had been in the habit of standing for many hours a day, resting her whole

weight upon the left leg. This naturally has produced in her a slight lumbar convexity to the right, and the rotation of the vertebræ necessarily associated with this has made the left renal recess shallow and the right one deep, reversing the ordinary conditions. Instead, therefore, of being an exception to the general rule, her case really confirms it.

One other case of movable kidney that has been recently in our wards I must just mention to you, as it is of exceptional interest from the point of view of diagnosis. The patient was a girl æt. 18, who was admitted for inflammation of the appendix. She had had many slight attacks, there was no suspicion of abscess, and the attacks were characterised rather by pain than by constitutional symptoms. There was no fever, but merely local pain and tenderness, which came on suddenly, usually after exercise, lasted a few hours, and then passed off, without apparently having any relation to the state of the bowels. In addition, the right kidney was freely movable, swinging downwards on its pedicle. When the appendix was exposed, it was found to be swollen and congested, but there were no peritoneal adhesions, or stricture, or faecal concretions. It was in what is sometimes spoken of as a state of catarrhal inflammation. Now this condition of the appendix is often associated, as it was in this case, with a movable right kidney, and operation is always advisable, for if the appendix is left, it is almost certain some day to become the seat of a more serious attack. As a matter of fact, even in these slight cases, septic organisms can always be found in the walls, but either they are deficient in number or in virulence, or the circumstances at the time are not sufficiently favourable to them to enable them to do much harm; but what may be the nature of the association between these two affections, whether one is dependent upon the other, or both are dependent upon the same cause, is very far from certain. It has been suggested that a movable kidney may press upon the mesenteric veins, and so lead to an attack of congestion. Without saying that this never happens, I am not prepared to admit that it was the case in this one. The association, however, is so common that I cannot think it a mere coincidence, and I strongly recommend you, in every case in which there have been repeated slight attacks of inflammation of the appendix, to carefully investigate the condition of the right kidney. Unfortunately, it usually happens that the appendix has already undergone such extensive changes that it is not sufficient merely to fix the kidney in its place: the appendix must, as in our patient, be removed as well.

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WITH SIR HUGH BEEVOR AT KING'S COLLEGE HOSPITAL.

February 12th, 1902.

DIPHTHERIA.

GENTLEMEN,—Diphtheria is a disease in which medicine has made great advances. In cases with both pharynx and larynx affected requiring tracheotomy, the death rate has diminished by one half and more, and in other cases the death rate has diminished very nearly one half. This is all owing to the antitoxin treatment. But such advance is only to be seen under conditions of promptitude in both diagnosis and treatment, for it has been shown in a report issued in 1897 that cases, when treated on the first and second day of the disease, suffered but a mortality of 6 per cent.; if not treated until the third day of the disease, 12 per cent.; and if not until the fourth day of the disease, 19 per cent. This report referred to 100 cases, in each instance of similar age periods—a precaution you must observe in reading any fever vital statistics.

The diagnosis is evidently not always a simple matter in diphtheria; no less than every tenth person recently admitted for diphtheria to a London fever hospital was found not to be suffering from the disease, tonsillitis being most frequently mistaken for it. In the face of the remarks previously made, such errors in diagnosis may well be excused; errors of omission will be of far graver import.

This disease may now be defined as a specific infectious disease characterised by a local lesion, which is further characterised by the presence of the Klebs-Loeffler bacillus.

But it is evident you must make your diagnosis and commence your treatment before you satisfy yourselves that the definition is complete. I mean a culture may be taken, and should be taken, and the report given by the bacteriologist upon a membranous sore throat, though it is not to the interest

of the patient that your action in treatment should await the return of the bacteriological report.

You will then administer antitoxin. The extent of the local lesion indicates roughly the quantity of poison that is being thrown into the system, the duration of the lesion tells of the time during which it has been entering, and immediately upon your diagnosis and consideration of these facts will you determine the dose of antitoxin that you will inject. Time having been proved to be so great a factor, it is at once evident that the case which is seen by you on the third day, or later, does not require to be treated by a somewhat expectant system of successive dosage, but, as Sidney Martin showed some years ago, by an immediate large dose of six or seven thousand units. A case early seen may be treated by two thousand units, to be repeated in accordance with the behaviour of the membrane.

A membrane that in situation extends further or appears elsewhere than the tonsil itself is at once significant of diphtheria. A membrane limited to the tonsil is suspicious by its extent and by its tenacious adherence.

You may well appreciate the difficulties of diagnosis when you consider the membranous sore throat to be seen in scarlet fever. Occasionally in scarlet fever a membranous exudation is seen early in the disease ; this is found not to be diphtheria—it requires no antitoxin. From time to time, in institutions where scarlet fever cases are gathered together, membranous sore throats appear in the course of convalescence. This membranous sore throat is diphtheria, and requires treatment by antitoxin.

Fortunately we have very often the assistance of other more or less striking symptoms to aid us in forming our opinion. These symptoms have their origin in the toxins of the local disease, which in the diphtheritic sore throat seem more evidently or more rapidly diffused into the blood and lymphatics than in other sore throats, even of an infective nature. However, these symptoms must always be considered in relation to the severity of the throat ailment, and might be expected to also occur in the rare event of other infective throat disease of equal toxicity.

First in frequency of these symptoms come the enlarged lymphatic glands of the neck.

Secondly, and less constant as an early symptom, is albuminuria.

Thirdly, the lassitude of the patient is often a conspicuous symptom.

It is to these symptoms, then, you will turn your attention when diphtheria is in the neighbourhood, and cases of sore throat come to you even without a membrane, and at other times where any appearance of membrane is present.

Symptoms.—I will now consider some of the symptoms and complications of diphtheria under the headings of the several physiological systems. The most practical order for considering these headings is the order of frequency of danger to life. The *respiratory system* comes first. Here we have to deal with membranous croup, which may be diagnosed at sight if, as is almost always the case, the fauces are also infected. If not, in children you may obtain a view of the epiglottis, but hardly of the larynx. But you may, by symptoms already mentioned, be sure that the affection of the larynx which the patient proclaims in voice or stridor is a membranous one. You may assist your judgment by process of exclusion. Membranous croup is the severest form of croup. In influenza epidemics laryngitis may be prevalent ; otherwise acute laryngitis in children is an affection which usually occurs as an exacerbation of a chronic laryngitis, and the child has suffered from previous attacks of laryngeal catarrh. Laryngismus stridulus occurs in small boys ; the “child crowing” at night, and its short duration afford you a diagnosis.

The danger in membranous croup is not only the imminent danger of blocking of the larynx, but danger by the very great local extension of the poison producing membrane, which in many cases extends down the trachea and larger bronchi. The first danger is evidently the greater the smaller the glottis or trachea, that is to say, the younger the child, for the cast of membrane may be of very similar thickness. Before the days of antitoxin it was a far less frequent occurrence that the patients should successfully expectorate these casts, even after tracheotomy, than is the case now. After antitoxin administration, not only is extension of the membrane, as may be judged from what we see on the pharynx, immediately checked, but rapid liquefaction sets in, and the casts are far more readily expectorated. However, these cases are still very dangerous, and pneumonia is a common complication.

Circulatory system.—Next in order of danger is the damage inflicted by the poison upon the heart. In almost every case, as a symptom we may note a remarkably low blood-pressure, namely, a very low tension of the pulse. Unlike voluntary muscle, automatic heart muscle is soon damaged by the poison. Histologically albuminous granules appear, or, at a later date, marked fatty degeneration is superadded. When the heart is affected more than usual in diphtheria, this can be more readily discovered by attentive auscultation than averted by treatment. We may hear at first "spacing" of sounds; it is this lower blood-pressure that delays the closure of the aortic valve so much that a longer interval than usual may be observed to elapse from first to second sound. Such may be taken as an auscultatory danger signal; it is followed later by rapid feeble sounds, in which the "spacing" is no longer remarkable. Death by cardiac paralysis may occur at any time from within the first week to late in convalescence.

Renal system.—I have mentioned albuminuria as a symptom of toxicity. A fatal anuria may supervene. At the autopsy such a case will show the kidney epithelium affected by destructive change, analogous to that of the cardiac muscles; we shall not see the signs of an acute nephritis. Thus the pathology is allied to that local necrosis of epithelial cells, a feature of the local lesion.

Nervous system.—The nervous complications, distressing and prolonged as they often are, remain fortunately of far less danger to life than those we have briefly discussed. Paralysis begins first of all, usually either at the palate or the eye. A nasal change of voice may be traced in the first before the patient suffers from regurgitation of food. Not only may there be strabismus, but the involuntary muscle is more likely to be affected, leading to dilatation of the pupil and loss of accommodation; these are very frequent occurrences. Such troubles may begin as early as the fourth day, and they have been recorded as late as the ninetieth day. In neuritis of the lower limbs, loss of knee-jerk is not an early symptom. A paralysis of the diaphragm is a complication liable to occur as a result of this diphtheritic poison, and, if intercostal muscles are also severely affected, with fatal result.

Before mentioning the treatment of symptoms

one may insist nothing can replace the want of that prompt attention to antitoxin administration, which I have already spoken of.

Symptomatic treatment.—Wide experience in former days has proved how little can be effected in the local treatment of that poison producing membrane. Still it is one point of possible attack. Its depth prevents ready penetration of any application, and demands that glycerine be an adjunct of any germicide we apply, for by glycerine we get those mechanical powers of endosmosis which we must believe to be of great assistance. Perchloride of iron was very usually employed with glycerine. This, it is well to remember, is efficient as an antiseptic. Sulphur has been credited with being of great service, and also paraffin. In treating respiratory complications, there is less occasion nowadays to have recourse to inhalation vapours and nebulæ. I have considered in former days that the patients derived more comfort from the nebulæ of sulphurous acid solution than from any other.

In view of tracheotomy profound cyanosis should not be allowed to develop before tracheotomy be performed. We have the less reason to hesitate seeing this operation saves so many more lives than before the use of antitoxin. Intubation is declared by Bosworth to be the more successful proceeding up to the age of five, and after that tracheotomy; you must remember that of the two intubation requires the more highly educated hand.

In striving to avoid those dangers which arise when the heart is especially affected, perhaps more is to be hoped by the employment of a drug such as arsenic, which is especially of service in promoting nutrition of cardiac muscle, than by the employment simply of brandy and other cardiac stimulants. It is important to give orders that the nursing of the patient be so conducted that no exertion be allowed, even during convalescence, until we are satisfied of the progress of the heart towards recovery. In dealing with threatened anuria, we may make any efforts to relieve the kidneys of their work, hoping thereby that such time will be gained as will give them recovery should the powers of the patient hold out; unfortunately, these powers are not such as to allow such vigorous measures of packing and cupping as we should usually employ in cases of acute ne-

phritis, though such would be indicated if possible.

Nerve complications, though often prolonged and sometimes distressing, admit of a good prognosis. After the acute stage of the disease is passed we must aid the recovery of that segmental neuritis upon which the symptoms depend by the use of the nerve-nutrient strychnine. The paralysis of palate must not prevent feeding, so nutrient enemata or feeding by catheter is not to be delayed.

Prevention is better than cure, and this disease particularly should not be passed by without reference to preventive treatment. As treatment is bound up with diagnosis and pathology, so preventive treatment is bound up with ætiology. A good example of the ætiology of the disease is seen in the occurrence of post-scarlatinal diphtheria which I have mentioned. There it is evident that a throat already weakened by previous inflammation is highly disposed to attack by Klebs-Loeffler bacillus. There it is also evident that the aggregation of patients whose throats are not healthy is usually the precursor of any such attack. Exposure to risk of infection from a previous case need not be considered a necessary precursor under these circumstances. Knowing that the bacillus of diphtheria is occasionally to be found in healthy throats, it is reasonable to suppose any of these unhealthy throats may acquire virulence without exposure to a previous case.

Preventive treatment, then, is by no means limited to isolation and disinfection, which are important here as in other diseases. Preventive treatment should be carried further, making an effort in the case of any prevalence of diphtheria to maintain the children healthy and free from sore throat. Two factors are very frequently met with which particularly undermine the health of the throat; these are dampness and impurity of air. Dampness of dwellings has been claimed as the predominant factor where diphtheria has persisted in one locality year after year. Evidence of this influence has been seen in a public school in a very healthy neighbourhood, where, after repeated epidemics, it was found necessary to deeply drain the whole subsoil of the playground around the school with effective result in the history of the institution. Impure air has seemed sometimes to be a potent influence in causing sore-throat by reason of ill-ventilated buildings, and in the case

of people exposed to sewer gas or to over-crowded rooms. It is not rare to hear of a case of diphtheria in brand new houses; probably the traps have been dry, and the house flooded with sewer gas, or the house damp. Aggregation of children, affecting ventilation, has much influence in schools.

Schools are an interesting problem in diphtheria, for school children are sufferers not only by reason of their age but also by reason of their attendance. Of course, if infection once starts, it can rapidly spread not only by proximity but by the rever-sionary habits of the school child, who may lick the slate to clean it, or pass a sweetmeat from mouth to mouth. The rise of diphtheria every year after the summer holidays is, then, something more than a seasonal phenomenon. By disallowing the attendance of children with unhealthy throats, prevention can do most to stamp out an epidemic at the opening of any school after holiday time in summer, or after being closed for a previous attack of diphtheria. Schools, then, are, let me repeat, a great source of infection, and it is not enough to remove the affected patients and shut up the schools, but when the schools re-open you want also, by a kindly arrangement with the authorities (who, I may warn you, are very shy if you attack them in a demanding manner), to inspect all the children and allow none to remain who have sore throats. We have learnt that there are in healthy throats micro-organisms which cannot be distinguished from Klebs-Loeffler bacillus, and the aggregation of two or three sore throats seems to allow of a regular outburst of diphtheria.

I. S. STONE, on closure of the abdominal incision, says the following procedure should be carried out:—(1) Remove all excess of peritoneal flap, also remove all loose pieces of muscle or fat, as these bits of tissue may become necrotic; (2) bring peritoneum, muscle, fascia, etc., together to meet similar tissues of the opposite side, without space between layers for collection of serum or blood, and yet without strangulation. The sutures to be placed in such manner as to make pressure as much upon the fascia, or tissue in the centre of the circle made by the suture, as at the peritoneal or skin surfaces. The interrupted sutures will answer these requirements in nearly every respect, the exceptions being its non-absorbability, and also its inability to bring like surfaces together accurately.—*Philadelphia Med. Journ.*, February 15th, 1902.

THREE CLINICAL LECTURES
ON
NERVOUS DISEASES IN
CHILDREN.

Delivered at the Medical Graduates' College and Poly-clinic.

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Eastern Hospital for Children.

LECTURE III.

GENTLEMEN,—I want to-day to mention, and not much more than mention, the other paralytic diseases in children, because I have a good many cases here which I think will interest you much more than any set lecture, and I have also some lantern slides to show you to illustrate what I have said in the last two lectures.

We were considering last time the diseases of the spinal cord which occur in children and cause paralysis, and we got as far as the consideration of myelitis from pressure, from spinal caries, or tumour of the cord, or from tumour of the meninges, which may also cause pressure and set up myelitis and consequent paralysis.

I have also to speak of spina bifida and syringomyelia. These are bracketed together because to some extent they both depend on a similar developmental defect in the spinal cord. Syringomyelia is probably the most common and the most important. Syringomyelia is a disease which shows itself very often not in very early life but I think most frequently in early adult life, although there must be presumed to be some defect in the development of the spinal cord, which usually shows itself in some wasting, similar to that in progressive muscular atrophy, the wasting usually affecting the small muscles of the hand, including those of the thenar and hypothenar eminences. Associated with this and distinguishing it from muscular atrophy is the defect of sensibility. Whereas a patient with syringomyelia may often be able to feel ordinary touches and to recognise them as touches, he or she is unable to appreciate thermal stimuli or the stimulus of cold or of pain, so that there is anæsthesia for pain and anæsthesia for temperature as a characteristic. Sometimes, when the condition has reached its

extreme degree, there is also anæsthesia for ordinary sensibility. It is a rare disease, but, of course, an interesting one.

I may also say a few words about disease affecting the white matter of the spinal cord. Of course the white matter is distributed in the posterior, anterior, and lateral columns, and we may get an affection of the posterior columns or of the lateral columns. The characteristic of the posterior columns is what is known as Friedreich's ataxy; and although the posterior columns are chiefly affected in that condition, one must recognise the fact that notwithstanding the disease falls with greatest force on the posterior columns, the anterior and lateral columns also suffer to a slighter degree. The change which occurs is one of sclerosis, and probably illustrates a disease in which, as I said at the commencement of my lectures, certain parts of the nervous system are badly developed and are prone to early death. In Friedreich's ataxy, from the morbid anatomy point of view, the disease is in the posterior columns, and more slightly in the lateral and anterior columns.

As regards the symptoms, the disease is characterised by difficulty the patient has in walking, he has what may be called a cerebellar reel, and there is usually present also a certain slight nystagmus. There is, however, no interference with the pupil. There is also frequently a lateral curvature and some deformity of the foot, which I shall show you in a patient to-day, and there is loss of the knee-jerk, and often slight nystagmus and defect in articulation, but no affection of sphincters. These are the prominent symptoms of Friedreich's ataxy. So much for disease affecting most markedly the posterior columns.

There is also a similar disease affecting the lateral columns, although it is only in comparatively recent times that this type of disease has been recognised. It is a form of lateral sclerosis which occurs in patients, usually in early adult life, who have previously been quite healthy, and who have had no difficulty in walking. Here, again, it seems as if we had to deal with a developmental disease, a disease, that is to say, in which there is some inherited weakness, but in such patients the weakness affects the pyramidal system and does not affect, as in Friedreich's ataxy, the posterior columns. Such a condition is very much like what you get as the result of birth palsy. The

difference consists in this: that in this inherited lateral sclerosis you have had a period of ordinary life, and no difficulty or disability in the way of walking, whereas in the patients who are the subjects of birth palsy or so-called cerebral spastic paralysis, you have a history that the patient has always walked badly, and is nearly always found to have been late in commencing to walk. I shall show you to-day a case of the cerebral spastic type. The other congenital spastic paralysis, so-called inherited spastic paralysis, is rather uncommon, and I have not been able to get one to show you. But so far as the patient goes his condition is simply one of spastic paralysis, and you arrive at the diagnosis by the fact that the patient has had a period of normal life, and by the fact that there is no other symptom present suggesting any other disease which is likely to cause spastic paralysis, and also the further fact that there is usually more than one member of the same family affected.

I might now just say a few words about the diseases of the peripheral structures. The conditions under which such structures are affected are chiefly diphtheritic paralysis, although in that we have probably to deal with an affection of the whole of the lower neuron; not only of the peripheral nerves but also the cells in which the axial fibres originate. Another condition is that which I referred to in speaking of anterior poliomyelitis. I said in connection with that that whereas in some cases you probably have an affection of the spinal cord alone, in other cases you have an affection of the spinal cord and of the peripheral nerves, those, namely, in which pain is a prominent feature. In still another I believe there is an affection of the peripheral nerves only. Those are the cases which, in their onset, are characterised by considerable paralysis, and which are also characterised by complete recovery. In my experience, when the anterior horns are affected, recovery is not absolutely complete, but I have seen one or two cases in which, with a history strongly suggestive of anterior poliomyelitis, and with symptoms very suggestive of that affection also, there has been complete recovery. Those, I believe, are cases in which only the peripheral nerves are affected, and their most prominent symptom, besides the weakness, is the severe pain in the limbs. So as a result of the same poison you may get either the

anterior horns affected or the peripheral nerves, or a simultaneous affection of both anterior horns and peripheral nerves.

I shall now only briefly refer to the muscular diseases, because I spoke about the myopathies a few days ago here. Those have been divided into the pseudo-hypertrophic type, the idiopathic type, and that in which the face is affected. But I think it is better to consider those diseases as being one, and that these are simply different varieties of the same disease, namely, myopathy, to distinguish it from myelopathy, in which the spinal cord is affected. The myopathies are distinguished by the fact that so far, by our present methods, we have not been able to discover any characteristic change in the spinal cord, so that the disease would seem to depend on some inherited weakness in the muscular fibres themselves, although further research may show changes in the cord. The diseases are characterised by great weakness. In the pseudo-hypertrophic type you have as a prominent feature the false hypertrophy of certain muscles, chiefly the calf muscles; in the idiopathic type there is a wasting of nearly all the muscles. But in cases which are apparently of idiopathic type you occasionally find a little pseudo-hypertrophy present. So it is an artificial distinction between the pseudo-hypertrophic and idiopathic types, and it is also a false distinction between those types on the one hand, and the type in which the face is affected on the other hand, because in certain cases in which the face is not affected at first, cases of the idiopathic or pseudo-hypertrophic kind, as the case may be, the face may be ultimately affected, and in the same family you may find one member suffering from pseudo-hypertrophic paralysis, and another member suffering from the idiopathic type of the same disease, and still another member of the family suffering from the type in which the face also is affected.

There has been lately described by Hoffman in Germany, and by Drs. Bruce and John Thomas in Edinburgh, another type of disease, to which I have already alluded, which seems to form a connecting link between the cases of myopathy and myelopathy, in which, with the changes in the muscles which are characteristic of pseudo-hypertrophic paralysis, are conjoined changes of a trophic character in the anterior horn-cells of the

spinal cord. So these cases, which are extremely rare, seem to form a connecting link between cases of myopathy and those in which the spinal cord is the chief part involved.

One very rare form of disease which I might allude to is the periodic paralysis, in which the patient, apparently perfectly well at other times, suddenly has an attack in which all the muscles of his body become extremely weak; and the curious thing is that in this disease not only do the muscles become weak, but they gradually lose the faradic contractility, and a patient who some hours before was able to walk about with perfect freedom suddenly becomes helpless, and not only has lost voluntary power, but the muscles will not respond to the faradic current. In the course of a few hours the paralysis usually passes off completely. I have only seen one case of that disease, and it certainly was a most striking condition. One could hardly realise it unless one saw the patient when he was well, and then saw him in one of these severe attacks. Its pathology is quite obscure.

I need only mention Thomsen's disease and conditions of myasthenia. Probably Thomsen's disease is one which might be described as myasthenia, and it is one in which the muscles apparently take on a strong tendency to spastic contraction, and as the patient sits he is unable at first to get up on account of the spastic condition of his muscles, but after making considerable efforts he is at last able to walk and to use his muscles, and as he walks and uses his muscles the freedom of movement becomes more and more pronounced. There is another form of myasthenia (Thomsen's disease is usually spoken of as myotonia), and that is myasthenia gravis, in which, without any appreciable or any discoverable lesion in the central nervous system, you get very great weakness of the structures which are served by the bulbar and spinal nerves. There is difficulty in swallowing and paralysis of ocular muscles, and in some cases death occurs. After death there is little or nothing to show in the central nervous system as the cause of the paralysis. These conditions are spoken of as myasthenia gravis because of the severe form which the disease takes. But seen clinically they are subject to great variations, and a patient whom you see in a very hopeless condition one day may be very much improved the next day. Besides this myas-

thenia gravis I am sure there is another form of myasthenia, that in which there is an inherited weakness of the muscles, in which the patient is unable to use his or her muscles for any long time, and they very quickly give out. I have not seen more than two of those cases myself, and I am not familiar with the disease. Those occurred in two members of the same family. They were to all appearance healthy and sound in every way, but after they had walked about one hundred yards or more, certainly a very short distance, there was a very great difficulty in moving the legs, and the gait was very manifestly affected. What the nature of that disease is I do not know, but it seems to me to be very closely analogous to those cases of myasthenia gravis, although one would say it is a disease of a much lighter form, because it apparently does not threaten life by involving vital structures, but is a source of the greatest possible inconvenience and disability to those who suffer from it.

I will now show you cases which I have brought. The first is a case of birth palsy. This child was born at the seventh month. Her walk is distinctly spastic. It has not become worse; in fact it is better than it was in some ways. She wears slight supports for the legs. She has exaggeration of reflexes. She has walked since she was six years of age. She is perfectly well in every other way, and she is intelligent, but is just now suffering from anæmia. She is left-handed, but the hands are perfectly good. The fact of her being left-handed might suggest that possibly the centre for the right arm had suffered at the same time that the legs suffered. It is one of the cases in which probably only the leg arcs have suffered, but it illustrates what you often find in those cases in which the legs only are affected, and in which they differ very materially from those in which all four limbs suffer, in that she is quite intelligent, and, indeed, is unusually bright, so that apparently her prefrontal areas have not suffered at all. The labour was a very long one, and it was a premature labour, so that all the conditions which you find in these characteristic cases were present. Her weakness and paralysis were recognised as soon as she was born.

This little boy illustrates the other form of cerebral disease in childhood, namely, the hemiplegic form. He was well until he was five years

of age, and then he had a series of fits which affected the left side. Ever since then he has been weak in this side. He is not able to use his arm, and he is still subject to fits on that side. As a rule, these hemiplegic cases are simply hemiplegic. He, however, has a peculiar condition of tremor in his hand, very like the tremor of disseminated sclerosis, in that it is evoked chiefly when he tries to touch anything. That tremor is spoken of as post-hemiplegic chorea.

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Eclampsia.—K. A. Herzfeld ('Cent. für Gyn.,' No. 40) summarises the pathological findings in eighty-one autopsies upon cases of eclampsia. All showed lesions of the uropoietic tract: chronic nephritis in 46.6 per cent.; parenchymatous degeneration, acute nephritis, etc., in 31.1 per cent.; bilateral compression of the ureters in 22.3 per cent. Kundrat has shown that this compression occurs only in primiparæ at an advanced period of pregnancy, and is found in cases in which an unusually high or low division of the common iliac artery causes the ureter to be pressed further forward than normally against the uterus. In thirty-two of the eighty-one cases hæmorrhagic hepatitis was found, and in four of these ureteral compression was also present. In twenty-eight, parenchymatous degeneration of liver and kidneys was observed, complicated in four instances by compression of the ureters. In twenty-one cases no macroscopical lesions of the liver were seen, and in ten of these the ureters were compressed. From these statistics Herzfeld concludes that the disposition to eclampsia usually arises from changes in the uropoietic system. He urges early and rapid delivery, and in primiparæ would favour Cæsarean section as the best means.—*American Journ. of Obstet.*, February, 1902.

MESSRS. BURROUGHS WELLCOME AND CO. have sent for notice "Tabloid" Mercury Iodide, Yellow, gr. $\frac{1}{4}$. It is a pure mercurous iodide and a definite chemical salt. It contains no free mercury, and is sufficiently stable if ordinary care be taken for its preservation. Therapeutic trials have demonstrated its reliability and efficiency.

A DISCUSSION

ON THE

TREATMENT OF INOPERABLE CANCER.*

MR. MCADAM ECCLES said that a case of carcinoma beyond treatment by operative measures was terrible and distressing, both to the surgeon and to the practitioner who had the case under treatment, and of course it went without saying that it was most terrible to the patient. Therefore, if there were any other than local means that could be taken, it was good that they should be discussed. He would take as an example but one of these terrible cases, which were, unfortunately, so frequent, of recurrent carcinoma of the mamma, or rather recurrent carcinoma after operations upon the mammary gland:—a woman presenting herself with visible recurrence on the chest wall; a primary, a secondary, or even a tertiary operation had possibly been performed, and yet there existed a recurrence, and there was in front of her eyes its steady progress. Anything that might remove the surface disease would be a relief to her mind, and death would be easier if it were due to hidden visceral deposit. There were quite a number of methods which were in the experimental stage at the present time with regard to the removal of surface carcinoma. X-ray influence had apparently been successful in a considerable number of cases, and in the treatment of recurrent carcinoma of the mammary gland there had come up of recent years an operation quite away from the local field of the original growth. It was removal of both ovaries, or double oöphorectomy. This operation had been followed by most happy results in a certain number of cases, while in others the result had been equally disappointing. Apparently at the present time in no given case could a surgeon say whether it was or was not going to be a success. He would allude very briefly to a successful case. It was that of a lady who had had a primary operation for carcinoma of the breast performed six months before. Within three months

* Abstract of a discussion held at the meeting of the West London Medico-Chirurgical Society, February 7th, 1902.

ferent nature to both the others. He shows signs of chronic ulceration of almost symmetrical form, and involving both cheeks and the bridge of the nose. Almost the whole extent of the nose is invaded, and reaching out to the malar eminence even to the front part of the ear. You will notice both ears are contracted, the natural roundness having disappeared; in fact, there is no lobule on either side. He has patches on the top of the head, where the skin is left absolutely denuded of hair. The scalp itself is thin; it is not simply alopecia, the whole of the scalp is thin, so that you can feel the bone just under the surface. It looks of the same appearance as the scar on the face. There is enlargement of the glands on the left side, especially the one opposite the bifurcation of the carotid. One, which seems to have retrogressed to a very small extent, can be distinguished in the submaxillary region. There are one or two to be felt under the sterno-mastoid on that side, so that there has been marked glandular involvement in this case. The question chiefly is, I think, Is it a case of lupus erythematosus, a case of true tuberculous lupus, or a syphilitic affection? It started on the ears of this patient twenty years ago, and he is now forty-two years of age. For a year or two it attacked the ears only, and in the winter time it was attributed to frost-bite. As a boy he says he was subject to severe chilblains. He has lived in England all his life, and has only been abroad three weeks, and that was not to a country where he is likely to have been frost-bitten. The original scar on the cheek was there for three or four years without anything else, then it started simultaneously on the bridge of the nose and the ear. The glands in the neck became enlarged immediately after he left school, at the age of fifteen. They broke of themselves. No one can doubt from the history and the date of the onset that he had tubercular disease of the glands of the neck. You know that the cheeks, ears, and backs of the hands and top of the scalp are the chief sites for lupus erythematosus, and the only point in which the distribution of these lesions does not conform to that is in the hands. Presumably he has not a good circulation, but he has outgrown the tendency to chilblains. The remarkable shrinking of the ears may have been due to lupus erythematosus, but there is no pitting in the concha. There is no reason to

suspect syphilis, and the question narrows itself into lupus erythematosus or ordinary lupus, and that, after all, is only of argumentative interest. The ingenious diagnosis has been made that it is an instance of "an erythematoïd form of lupus vulgaris." The connection between lupus erythematosus and tubercle is very close, and you can get more information as to inherited tendency to tubercle in the subjects of lupus erythematosus than even in true lupus, as my father has pointed out. Here there is the coincidence between the condition you see and the breaking down of the glands of which we have had a history. I have had occasionally to scrape out glands or excise them where patients have suffered from lupus on the part of skin drained by those lymphatics, but it is certainly rare. This is not a case for heroic treatment, and it is difficult to attribute recovery in cases of this kind to any one drug which may be given. I know that Dr. Payne has drawn attention to striking cases of complete recovery from lupus erythematosus after long courses of quinine, but other observers have recorded recovery from long courses of cod liver oil and other drugs. It is probably most important that the patients with this condition should be kept in as good health as possible. This is not a case for cauterisation or excision or scraping. We must allow it to die out. On some parts, such as the scalp, you will notice it has already died out. Dr. Hichens treated it vigorously with carbolic acid, and for two months previous to that he was in St. Thomas's Hospital. Seven years ago the surface was one erythematous mass, the patient says, almost as thick as his hand. One dermatologist treated him with aniline dyes, but it had no effect. There is very little active disease left now. Of course carbolic acid is the mildest of the superficial forms of cauterisation, and it is interesting to hear that it has done some good.

With regard to the light treatment, if the man chose to go to the inconvenience and was patient enough to submit to that treatment there would be no harm in trying it, but we are bound to tell him it would be a very slow process, and even then one could not definitely promise anything like a cure. In fact, one must admit that the results of the light treatment for lupus erythematosus have been hitherto discouraging, both in Copenhagen and London.

A SYSTEM OF RADIOGRAPHY.*

By E. W. H. SHENTON, M.R.C.S., L.R.C.P.,
Radiographer to Guy's Hospital.

LADIES AND GENTLEMEN,—In all branches of medicine and surgery we learn a system of examining our patients. Such systems are the outcome of monotonous clinical work. In radiography the methods are at present *dilettante*, and if we are united in thinking that this diagnostic method is to be of every-day use, we must gather up the reins and go forward in a more orderly manner.

Let us consider first the end we have in view, and then select the most efficient means to attain

taught that to listen to a chest while in a strained position is to run the risk of missing details from inability to entirely concentrate the mind. From this point of view, then, we are justified in considering our own comfort a little. Now let us apply these conclusions to the arrangement of the patient who is to be examined, considering carefully the best relative positions of patient, operator, and tube. The best arrangement will be that by which the best screen effects are obtained. I wish here to state as an axiom that careful screening is the scientific basis of radiographic examination. The man who pretends to give a radiographic diagnosis, and relies merely upon making a sandwich of his patient between the plate and the tube,

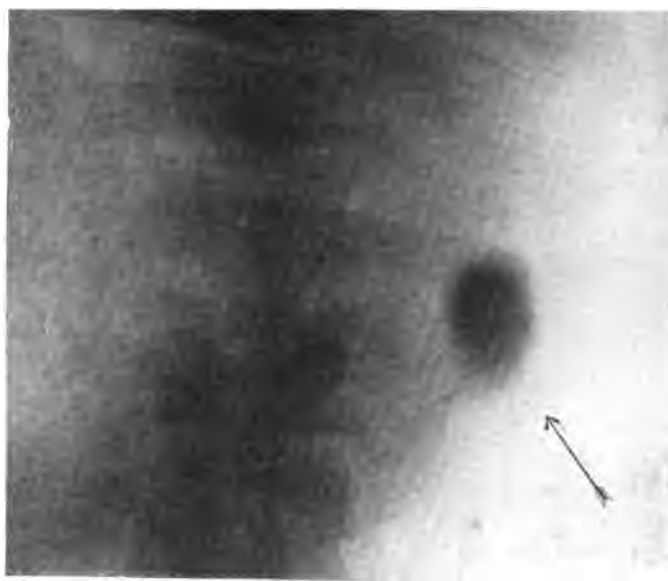


FIG. 1.—A large calculus in the right kidney, composed of oxalates and phosphates. Exposure 20 secs.

this end. Undoubtedly we are all striving to benefit our patient, directly by the application of the rays to his person, indirectly by adding to medical knowledge. In getting the utmost benefit out of radiography for the patient, we must not forget his personal comfort, nor the elimination of risk, while throwing scientific light upon a disease. The examination should be as short as is consistent with thorough investigation. An indirect bearing upon the welfare of the patient is the comfort of the operator. We have been

* Delivered before the Röntgen Society, February 6th, 1902.

is on a par with him who trusts to one single method of clinical examination, say, for example, auscultation. He may become very clever with it; he may have few failures, but however brilliant he may be, he will assuredly be outstripped in the long run by the man with fewer brains, who methodically goes through the routine system he has been taught. There is also another important point bearing upon the subject of screening. Any one can be taught to place a plate beneath an injured limb, to adjust a tube above it, and with a small knowledge of photography to produce a radiogram. If such work as this is to be accepted as

the scientific use of the Röntgen rays in medicine, then there is no such thing as radiographic diagnosis. No man, however anatomically or radiographically educated, has a right to give an opinion merely from a radiogram. It cannot be denied that he is often correct, even as a man may judge from the outside of a house what the inside is like, but he relies upon the element of chance, and this is an element we all try to exclude from sound diagnosis. These, taken with many other facts, tend to support the claim made for the value of screen work from the patient's point of view.

We will return to the consideration of patient, operator, and tube. We will assume that most patients that seek the aid of the X rays are more comfortable lying down than standing up. Perhaps you may object to this? If so, I will put the matter at its lowest estimate, and say one half prefer lying down. So much for the patient. Now for the operator. Is he more comfortable when the patient is vertical, or when he is horizontal? Those who have had a little routine experience will not hesitate to decide this question in favour of the horizontal. When the patient is vertical the operator must hold the screen in his hand unless he have some contrivance for holding it. When the patient is horizontal the screen is laid upon him, and the force of gravity replaces apparatus. When the patient stands before an X-ray tube, the only part that the operator examines in an unstrained position is the upper part of the thorax. When he is horizontal all parts of the body are equally accessible. From the operator's point of view, then, the horizontal position is the more convenient one. The abdominal and gluteal regions present the greatest opacity to the X rays; anything that can be done to lessen antero-posterior measurements will facilitate the examination of these parts. There is considerable reduction when the patient is in the horizontal position. A patient standing before a tube may inadvertently come into too close proximity with it, whereas, when lying in a horizontal position, this cannot happen.

To come to more concise details, I wish to show you this couch, which I designed some years ago, and which is now in use at several London and provincial hospitals, as well as in Paris. The essentials are a table with a canvas top and a tube-holder of a common-sense pattern that supports

the tube at both ends. I would specially call your attention to this tube-holder as one of the most practicable comforts imaginable in an X-ray sense; it is so arranged that by means of simple mechanism the operator moves the tube into any position while he watches the effect upon the screen. A very small, but very essential, detail is how best to connect the tube with the coil, so that it may move from end to end of the couch with perfect freedom, and yet without a slackening of its leads, which would, of course, cause leakage and sparking. I am sure it is unnecessary for me to point out the utter futility of trying to insulate leads from the secondary with any other material than atmospheric air. Fine hardened brass wire is moulded into a long tension spring by firmly binding it round a half-inch cylindrical rod. This, when connecting tube with coil, will allow of considerable extension, returning to its original size when required. In other words, it gathers up its slack as the tube nears the coil.

From time to time apparatus have been designed, no doubt with a view to making radiography easy to the uninitiated. A great deal of the want of value in these contrivances is due to the fact that the inventors have been theorists, and not everyday workers in the realm of X-ray diagnosis. The result is that although radiography is a young science it is already entangled in a maze of complications. It would be unfair, perhaps, to call attention to any one single piece of apparatus, for in judging these we must remember that our actual quantity of Röntgen light has been steadily augmented, and the conditions under which these things may have been useful have now changed. In surgery we have seen the same accumulation of complicated accessories, and if one takes the trouble to wade through a list of all the instruments that might be used one is impressed with the fact that the simplest are those that survive. I would suggest that the greater quantity of Röntgen light we obtain, the simpler will be our apparatus, for were our light so bad that we could not screen, everything would be of necessity photographic, and it is usually the photography that elaborates our apparatus. I would therefore point out that everything that tends to increase the quantity of the Röntgen light tends to simplify our instruments. This is one, though not the most important reason for striving after quantity of

X-ray light, and this seems a fitting opportunity to speak of tubes. There is a quality in a tube known as definition, and let us consider what the



FIG. 2.—A phosphatic calculus in the left kidney.
Exposure 18 secs.

value of this quality is. In speaking on this matter I do so from the point of view of the practical every-day and all-day worker, and I hope, therefore, that you will carefully consider the following facts: How is definition secured in the ordinary photography of animate objects? You will say by using finely constructed lenses. Yes, but is a finely defining lens in the case where a lengthy exposure of a moving object is given, say, for example, where the light is very bad, of any avail? I think you will agree with me that better definition can be got with a half-penny magnifying glass and a drop shutter in bright sunlight than with a valuable lens in the half dark. Let us apply this parallel to radiography. The tube that gives fine definition but requires to be illuminated for a considerable period before it impresses the photographic plate, will not give as good a picture as the tube with less defining properties but a greater quantity of light, for the chances of the part under examination moving are very much augmented by a lengthy exposure. We see, therefore, that shortness of

exposure is quite as essential for definition as defining quality in the tube. Therefore, if in a defining tube we are losing quantity of light, we have lost as much as we have gained. But we have lost more than we have gained, for we have lost screen effect, and have had to give prolonged exposure to get the same picture. Prolonged exposure means possible injury to the patient, and certain discomfort to patient and operator, so that, again, as we saw from the point of view of simplicity of apparatus, we see from the operator's and patient's points of view the desirability of increase in quantity of the X rays. I am assuming, hypothetically, there is such a quality as definition in a tube. I admit, theoretically, there is such a thing, but in practice I deny it. Take the best defining tube you can get and photograph with it a piece of dry bone and a piece of wire. Take the worst defining tube you can obtain commercially and do the same, and then state your opinion of the superiority of the first, and whether you consider it worth while sacrificing any advantage to this chimerical quality. In how many cases in our



FIG. 3.—Oxalate of calcium calculus in the right kidney.
Exposure 20 secs.

daily routine do we require minute definition to any considerable extent? I have estimated it at

perhaps 1 per cent. In how many cases daily do we require more light? The answer to this is simple, in every case we want more light. Whether it be for a needle in the hand, a stone in the kidney, or a fracture, we could always do with four times the quantity that is at present available.

You see before you to-night radiograms taken in the routine work of a London hospital. There is not a single instance in which the exposure exceeded a minute. The tube used was made from my suggestions by Mr. Dean, with many modifications that his skill and experience suggested. All ideas of definition have been sacrificed to the all-important one of increase in light. Do you consider that there are many patients to whom these radiograms apply who would have been benefited by greater definition? On the other hand, I believe that by shortening the exposures the chance of dermatitis is eliminated. In some thousands of cases in the last two years there has not been a single instance of skin trouble. This leads one to suppose that dermatitis is a preventable accident, and is indirectly prevented by the increase of light.

There is one complete class in which increase of light has been of first importance. I refer to cases of foreign bodies embedded in the tissues. Let us for the time forget the expression "localising apparatus," and ask ourselves what we require to do in this class of case. You will agree that we wish by one means or another to rid the patient of his trouble. If a friend had a buried treasure, and wished to give sufficient particulars to enable you to dig it up, would you prefer that he gave you a photograph of the field with the position of the treasure marked upon the photograph, or that he took you to the spot, and marking the ground, said, at such a distance immediately beneath this mark lies the treasure? No practical man could hesitate to choose the latter, for the best that could happen in the first case would be that you yourself would transfer the mark from the photograph to the field, and supposing you did this with great exactness, you would be no better off than if your friend with full knowledge had marked the spot for you. Now let us apply this to the finding of an embedded bullet that represents the treasure. Your friend in this case being the X rays, he, the X-ray friend, can either give you a photograph of the

whereabouts of this bullet, or he can mark the spot immediately above it. Thus, at the outset, by simplifying we are bound to be more accurate. Now I maintain that if a mark is made upon the skin just above the object sought, and if the exact depth of the latter is given, human aid can go no further.

Just a word upon the making of this mark upon the skin surface. Let the part to be operated upon lie in the position in which it will lie at the operation. Next use a large screen, and arrange your tube so that the bullet lies in the brightest part of the field. Holding a probe, pass your hand beneath the screen, placing the probe's tip against the shadow of the foreign body, turn up the light, and mark this spot on the skin. If the foreign body be a needle mark each end separately. Now this is the first step, and the most important in all localising operations.

How to find the depth from this point must now be considered. Take a very difficult example: the localisation of a small piece of lead in the neighbourhood of the hip-joint. You have made your mark upon the skin immediately over the bullet.

Now to make a digression. As we sit in a railway train the objects without appear to pass the window at varying rates, according to their distance away. The telegraph posts pass with great rapidity and regularity, trees in a near field more slowly, trees on the horizon more slowly still. Experience has taught us that those things that are nearer appear to pass more quickly than those more remote, and that objects that pass at the same rate are equidistant from us.

Remembering these facts, let us go back to the hip-joint we are examining upon the screen and, while looking carefully at the piece of lead, move the tube slowly to and fro. It is at once seen that the shadows of various objects present are moving at different rates, and as in the case of the train it was noted how the relative rates gave relative distances, so you note in this case that the faster the shadow of an object moves, the farther must that object causing the shadow be away from the screen, and that where two objects move at the same rate they are the same distance from the screen, that is, they are upon the same plane. Now for the first time we can form a rough idea of the relation of the foreign body to the bones, according to

whether it moves faster, slower, or at the same rate as they do, and it is surprising how a little practice will enable one to give an approximate measurement that will often not be improved on when exact measurements are taken. While the tube is being moved to and fro, as above described, take a probe in your hand and so adjust it that shadows of probe and bullet move at the same rate. It does not matter how near the probe is to the bullet so long as it is in the same plane. If you cannot trust your own judgment in this matter, mark the position of these shadows upon the screen when the tube has moved to its limit in one direction, and mark again when it has moved to its limit in

place of the hand and probe, and gives a reading in centimètres and millimètres. Of course, this whole process may be repeated from as many points of view as is desired. This completes the localisation in what I believe to be one of the most difficult of cases, but one which I have come across many times and in which I have never had occasion to make use of photography. Bullets in the hip region are met with occasionally; but needles in the hands and feet every day, and in these cases some modification of this method is necessary. Those of you who come across much of this work, and are in the habit of operating yourselves, will agree with me that

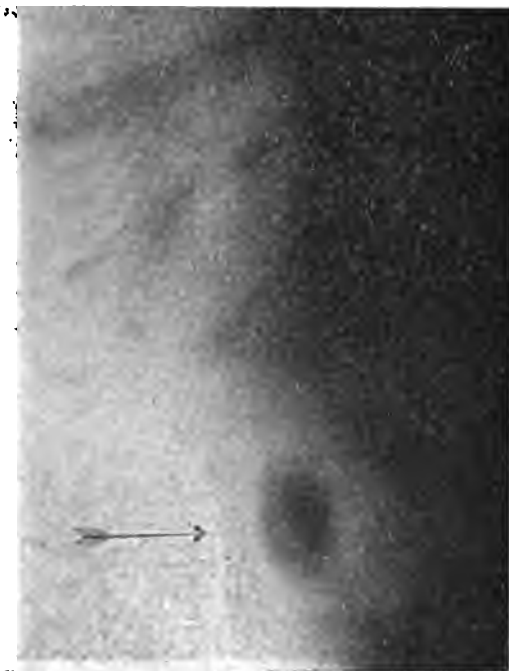


FIG. 4.—Seven calculi in the left kidney composed of phosphates and urates. Exposure 30 secs.

the other. If the first two marks are at the same distance apart as the second two, then your probe is at the same distance from the screen as the bullet, and since you can easily ascertain the distance of the probe from the screen, you have found the depth of the bullet from the mark on the skin.

You will no doubt suggest that inaccuracies may occur by shifting the screen, etc. To guard against these I have a small instrument which is screwed to the side of the screen, and takes the

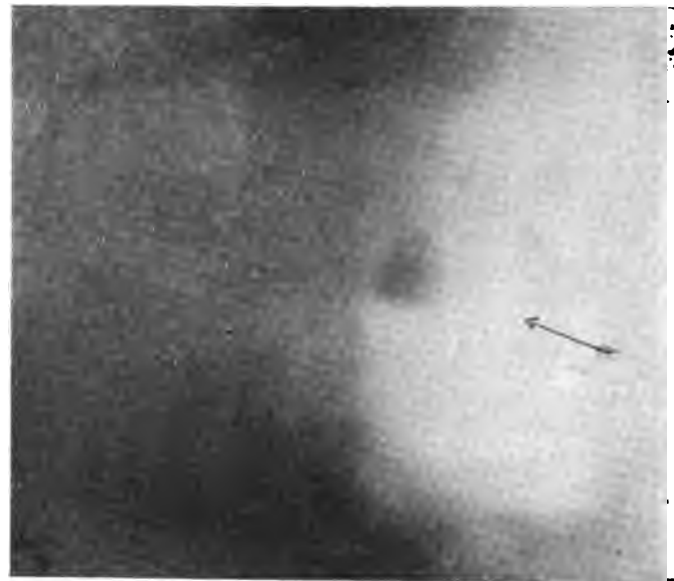


FIG. 5.—A calculus in the right kidney composed of uric acid. Exposure 30 secs.

mathematical accuracy is not essential to success. The following is a method that is used daily at Guy's Hospital:—Having marked the position upon the skin as described above, say in the case of a needle embedded in the hand, place the tip of a probe upon one end of the line marked upon the skin, and while held carefully in this position rotate the hand at right angles so that the thumb is uppermost. The probe is provided with a metal sleeve (for simplicity's sake wrap a piece of lead

foil round the probe), and so adjust this sleeve of lead foil that its distance from the probe's tip is equal upon the screen to the distance of the probe's tip from the end of the needle. Again, if you cannot trust your eye measure upon the screen. This will give you the depth of the particular end of the needle you are seeking. The process must be repeated for the other end. With slight modifications there is no foreign body capable of demonstration with the rays upon which the surgeon is justified in operating that cannot be accurately located in an inconsiderable space of time. I will make one exception, and that is in cases of foreign bodies in the eye, where, obviously, the situation is so different that it is unnecessary for me to point out the value of Dr. Mackenzie Davidson's work in this branch.

The point which I wish to bring before you is that we waste our time designing apparatus, if, by this system, data can be furnished which will make the process of extraction an accomplished fact in the hands of anyone who has sufficient skill to justify his operating upon living tissues; the said process being carried out without special apparatus, without the aid of photography, and in a space of time so short as to be negligible. I do not ask you to accept what I have shown you without sufficient proof, and this I am fortunately able to give you. In a whole year of cases in the radiographic department at Guy's Hospital, there was only one patient who left the hospital with the foreign body *in situ*; in all the other cases removal was accomplished easily and at once. The one exception was in the case of a patient who lived in the country and would not stop to be properly treated. This is not all; cases that two years ago would have been submitted to general anæsthesia have, with few exceptions, been operated upon while under the effects of eucaïn. Before leaving this subject I should mention that our screens are covered by transparent celluloid, which keeps the salted surfaces clean, and allows of marks being made upon it with a dermatographic pencil.

A large and important class of cases to which a system of radiography must adapt itself is fractures, their *detection*, their *exclusion*, and their *treatment*. We must consider *exclusion* first, as being at once one of the most useful and most difficult tasks that the rays are called upon to perform. Varied experience and varied success in the examination of

fracture has led me to formulate this rule as a safeguard to myself as well as to the patient: "Always photograph if you cannot see a fracture." One reason for this is that a fracture in perfect position can only be detected by the derangement of cancellous tissue, and the screen surfaces are too coarse for a proper appreciation of this. Of all uses of photography in Röntgen ray work I would put this first. There is no easier, perhaps no more dangerous, and certainly no more humiliating mistake that one can make than to guarantee the integrity of a bone that is unsound. There is no doubt that a man who has studied the subject carefully can put himself in a position to say definitely in doubtful cases of fracture of the bones of the limbs, "There is no fracture present," and the assistance afforded the surgeon by such an absolute statement cannot be over-estimated. The man who has educated himself to this point to speak positively and fearlessly, with a full knowledge of his responsibility, does much for the art of radiography, for he has proved that in this one instance alone radiography has superseded every other known method. And then, again, he rouses confidence in X-ray methods among his medical brethren.

As to *determining the presence* of a fracture, in many cases this is quite easy, and when a fracture can be seen upon the screen there is seldom need to go further, save in shoulder and hip and other obscure regions. In cases where there is difficulty in determining the presence of fracture, I have found the careful examination of the light area which represents the medullary canal to be of great value; want in continuity of this light streak invariably means fracture. Another suspicious condition is a seeming want of drawing, if I may use the expression, in the bone. If the shape of the bone strikes one as being unsatisfactory, especially when compared with the one on the opposite side, fracture is generally present. There is one condition that one must be prepared for, viz. a large extravasation of blood, which very much mars, and sometimes entirely obliterates, the image of the bone. But this condition need not deceive, for it is pathognomonic of fracture, and the case should be returned as such, and examined again at a later date. I had an example of this recently, a fracture of the lower end of the femur. The part was much swollen and resisted the investigation of the

rays. Later the limb had to be amputated on account of severe laceration of the big vessels by the fragments of the fractured femur.

Unquestionably the most valuable use of the

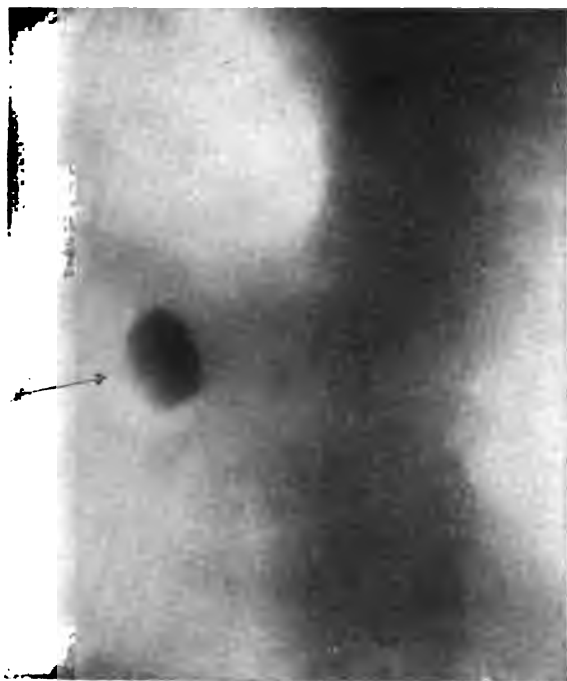


FIG. 6.—A calculus in the left kidney, situated immediately below a distended stomach. Exposure 20 secs.

rays in the abdominal region is in the detection of urinary calculus. I will describe the method of examining for this foreign body. Use the screen thoroughly, first from the front, then from the back. Any dark area in the renal region that catches your eye as you move the tube to and fro observe most carefully. It is no uncommon thing to see the stone upon the screen. I have done this myself in eleven cases in the past eighteen months. In photographing this region let the patient lie upon his face, his arms above his head, so that he cannot support himself at all by his arms. Move the tube until your picture upon the screen satisfies you, or until any suspicious object comes well into view, lay your plate across the renal region, and expose. Do not give more than one minute; with a medium-sized adult thirty seconds will suffice. It is very desirable to get a thin negative in these cases; great contrast may be pretty, but often defeats the purpose for which the examination has been undertaken.

The pelvis is another region to need consideration, and mostly we examine this to ascertain the presence of ureteric or vesical calculi, deformity, and fracture. Again the horizontal position will be found the more convenient. Examination must be made from the front and from the back. Difficulty will be experienced in photographing from the front, on account of respiratory movements, and in screening and photographing from the back pressure should be made upon the screen or plate to lessen the thickness of the gluteal region. It is surprising with a little pressure how well illuminated the pelvic canal becomes. All regions of the body may be photographed in the same manner by merely laying the plate where the screen has been. Adjust your picture upon the screen, a feat you can perform with great ease, as one hand controls the position of the tube, place the plate beneath the screen and leave the holding in this position



FIG. 7.—Example of a skiagram strongly supporting the negative evidence obtained by screening. Exposure 15 secs. In this the colon is shown by the light areas on either side, the stomach by the light oval patch at the left-hand top corner of the picture. The dark patches on either side of the spine represent kidneys which are only evident because of the flatulent distension of the intestine.

to gravity. During exposure you can thus see the picture that will be upon the plate. Of photo-

graphic methods it is only necessary to say, let the development be long, the negatives thin, and the printing paper brilliant.

If, in my endeavours to describe to you the system that we are using to-day at Guy's Hospital, I have failed to convince you of its efficiency, rapidity, and freedom from risk, I will ask you to believe that this is due to my lack of descriptive power, for that it possesses these essentials I am absolutely convinced. I would also claim your indulgence when you criticise the work you see before you by asking you to remember that perhaps in many cases prettier results could have been obtained had it not been our aim to minimise every risk to the patient by diminishing the length of time he is under the mysterious influence of the X rays. Furthermore, I would earnestly ask you to consider the desirability of introducing greater simplicity into methods and apparatus. Perhaps the value of simplicity at a large London hospital is not very apparent: but turn your thoughts for a moment to a South African battlefield, where everything must be sacrificed to portability. The requirements in London are a good screen effect and a darkened room; I have not been to South Africa, but I presume that darkness there possesses much the same quality as it does in England. What provision was made for the obtaining of a good screen effect? I can answer these questions by quoting from two very excellent radiographers who have been out there. Writing home, one says, "Here I have to give four minutes' exposure for the palm of the hand." Another says, "In every case of foreign body I have taken three negatives."

Read the list of apparatus I have detailed to you and read a War Office list of what were considered essentials for radiographic work, and draw your own conclusions.

Of the vital importance of rapidity of exposure with its accompanying screen effects there can be little doubt. A radiographic system of the present day should reckon its exposures in seconds only. If such a system involved great complications it would still assert its superiority on the grounds of economy of time and perfect safety, but, fortunately in this matter, as in most others, the simplest is the best.

[For the use of the foregoing blocks we are indebted to the Editors of the 'Guy's Hospital Reports.']

MEDICAL ANNUAL YEAR-BOOK FOR 1902.—We have received from Messrs. John Wright and Co., of Bristol, a copy of the 'Medical Annual' for 1902. This is the twentieth appearance of this Year-book of Treatment and Practitioners' Index, and it thoroughly deserves the reputation it has gained. The publishers must be congratulated on the success of their energetic efforts to supply medical men with accurate information concerning the latest questions of medical and surgical interest. The practical and useful character of this annual will be recognised by the perusal of the following passage:

"TREATMENT OF TYPHOID FEVER.—A patient seen in the very earliest stage of the disease, when diagnosis is still doubtful, should be confined to bed. The usual symptoms at this stage are persistent frontal headache, pyrexia, and *malaise*; and whether or not there be any departure from the normal action of the bowels, no harm can be done by administering a mild purge. The diet should consist of milk and water, beef-tea, soup, etc. Usually the patient is disinclined for food. When the disease is more fully advanced one of the most important questions is that of diet, all the more important in this affection because of the bowel lesion. One has to consider two points: the patient's capability of digesting his food, and the possibility of damage to the bowel by any particular article of diet; the first point is the most important since the latter depends upon it. Now, in order to ascertain whether food is being digested properly, it is absolutely essential to inspect the patient's stools at least once in the twenty-four hours, a point rightly insisted upon by F. J. Smith in an admirable paper on this subject.* If, the patient being on a milk diet, as is usual during the febrile period, curds are being passed, then that is an indication for further diluting the milk and giving less of it. Smith recommends, when undigested food is seen in the stools, the absolute prohibition of all food whatever for twenty-four hours. But the writer thinks it better to alter the food, either by dilution or predigestion (peptonising). We have said that milk is the usual diet during the febrile period and for some considerable time after. It should never be given pure, but diluted with water. The yolks of three or four eggs may be given in the course of the day, beaten up with the milk or made into custard and broken up. Beef-tea, chicken broth, and essence of meat may also be given."

* Lancet, February 2nd, 1901.

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* Specially reported for The Clinical Journal. Revised by the Author.

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A CLINICAL LECTURE

ON

- (1) **A FATAL CASE OF ACUTE (DOUBLE) PLEURISY ;**
(2) **SCHÖNLEIN'S DISEASE (ERYTHEMA MULTIFORME).**

Delivered at St. Bartholomew's Hospital,
February 28th, 1902.

By SIR DYCE DUCKWORTH, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—Before I pass to the subject of my lecture to-day, I want to call your attention to a very sad case which has occurred in my female ward, because it illustrates one of a kind which is happily not frequently met with, and which has some lessons of importance to teach us. It is the case of a nurse whom many of you may remember has been at work in Matthew Ward. She was on duty so lately as Sunday last, when she felt ill, and complained of shivering, vomiting, and general unfitness for work. She was ordered immediately to bed in John Ward, and was found to be suffering from severe pain in the left side, with feverishness, and was evidently very ill. I saw her on Monday, when she had manifestly symptoms of pleurisy affecting the left side, with some effusion. But she had a good deal of pain, and exhibited symptoms which showed her to be more ill than the physical signs alone explained. Next day she was more ill and depressed, and was evidently suffering very much pain, which had only been partially relieved by the application of leeches to the left side. On that day she began to complain of pain in the right side of her chest. There were then signs of involvement of the pleura on the right side, so that she had at this time pleurisy on both sides of the chest. Each day she grew more ill, and this brings us to Wednesday. On Thursday she was worse still, her pulse having increased in frequency, her temperature still remaining high, and the application of leeches not

having done very much to relieve the pain and effusion, which were evidently increasing on the left side upwards from below. Altogether the patient was exceedingly ill. This morning, soon after four o'clock, when I was summoned to see her, she died. Here, then, you have a case of a perfectly healthy and vigorous young woman, æt. 34, in the very prime of life, and yet in six days she has been carried away by an acute illness. It is an unusual thing for an acute pleurisy to prove fatal, that is to say, one presenting the physical signs which were present in this case. There was no great amount of effusion on either side, there was nothing mechanical to obstruct her breathing to any considerable extent, and it is therefore borne in upon us that this is a case in which some grave fault, such as an infection, was at work, which led to the production of these symptoms. Pleurisy itself is but a symptom. Fifty years ago physicians were content to label a disease such as we have had before us in this nurse simply pleurisy, and no more would be thought of it. It was looked upon as a disease in itself; whereas, now, in the light of more modern knowledge, we only regard pleurisy,—an inflammatory state of the pleura with more or less exudation, either of serum or of lymph, or of both, as a symptom of some underlying state of the system. In fact, it is to be regarded either as a result of invasion of tubercle on the one hand, or of some peccant matter, such as the pneumococcus, staphylococcus, or streptococcus, and to these toxic agencies I think we may add that of influenza.* Under the influence of one of these agents pleurisy is set up as a symptom. In this case which I have been relating to you I think one cannot but believe that it arose in some infection, and we look about to see what this could have been in such a case. We cannot find anything very remarkable. It is true that there was a case of influenza of a bad type in the ward a few days ago, and considering that influenza is again prevalent, it is not at all unlikely that this may have

been altogether a result of influenza, and, if so, it is a very grave and lamentable one. The fatal issue was not the result of the mere process in the lungs, but of some overwhelming dose of toxin, of which this pleurisy was the symptom. That is the impression we are to gather, because the case is a rare one, and it is of that description which has been called by the Parisian physicians fulminating pleurisy. We meet with fulminating pneumonia in which a person is rapidly struck down and destroyed, as by malignant small-pox, measles, or scarlet fever, conditions in which the patient is destroyed before the disease has had time to develop its ordinary symptoms. That is the light in which we must regard this case. We can only regret that it should have fallen on such a useful and honourable person as this nurse. Those who worked with her held her in great regard, and knew her to be a most conscientious and devoted woman.

To-day I wish to call your attention to a patient who has been nearly three months in Matthew Ward. He is a pianoforte maker æt. 31, and his history is very simple. He had been ailing on November 27th, 1901, having had pains in his knees which he called "rheumatics." A fortnight before these pains came on he suffered from sore throat. Two or three days afterwards the pains left the knees and "went to his stomach." (It is very important to follow out the language and ideas of patients as far as possible; you cannot do better. By the stomach, of course, these patients mean the abdomen.) With these came sickness and vomiting. The pain was not worse after food, but when he was sick soon after food the pain was relieved. A few days after his admission he suffered from stiffness in different limbs, and vomiting was occasionally present. The next thing we noticed was that a rash or eruption came out on the backs of his hands. This eruption was quite peculiar, and was exactly symmetrical. I can give you no better idea of the appearance than by handing round this water-colour drawing which has been excellently done by Dr. Picton. The rash you observe specially affects the extensor surfaces of the metacarpo-phalangeal joints, and on each of these places there was a red, raised, glossy patch with hæmorrhage and exudation into it, forming a rash which those versed in skin diseases would

* A small amount of slightly blood-stained fluid which had been removed from the left pleura was examined by Dr. Drysdale. He found pneumococci in cultures from it, which proved virulent on subsequent inoculation. It is not impossible that influenzal agency modified the course of the illness in this case, notwithstanding the above revelation.

recognise as a form of erythema. Together with that were some other spots in various parts of the body, more of a purpuric nature. The man was obviously ill at this time; the vomiting was very severe, and he brought up large quantities of bilious matter. Of course, a patient who has been three months in hospital provides material for very copious notes, which it would be tedious to read in a clinical lecture. We can summarise them by saying that the patient did not derive benefit from the remedies of many kinds which were tried, but from time to time fresh outbursts of rash in the hands, arms, and trunk, and especially on the legs, feet, and ankles, occurred, the skin lesions being mostly of a purpuric nature. Sometimes he was better and at other times worse. When his knees were bad his temperature rose, as you will see by the chart. Sometimes it went up to 101° , and on one occasion it was 101.4° , but never higher than that. Since that rise there has been nothing remarkable about his temperature, and for many weeks it has been almost normal. The man has remained unwell, with occasional vomiting, then getting a little better. Then the pains return, and following them the eruption reappears. He is still developing these purpuric patches. I have had him brought down for you to see. The purpuric patches which were on his hands have long since healed, without leaving any very recognisable scars. The legs present the appearance of an ordinary case of purpura; it is a maculated rash looking like the eruption of typhus fever, which, happily, is now practically an extinct disease among us. Over the patella, just as over the surface of the metacarpo-phalangeal joint, are lesions of this erythematous quality. The same are apt to occur on the elbows. The places affected on the backs of the knuckles still remain scarred. In one place there are slight erythematous lesions which may be called erythema papulatum. They occur over prominences and upon the extensor surfaces. Those are the main features of this case. His general health has improved a good deal, but he still has this purpuric tendency. As is often the case in purpura, the erect posture has a bad effect in inducing fresh outbursts. The patient will often show no signs of purpura as long as he keeps in bed. If he is allowed to sit up for an hour or two, he shows the purpuric spots next morning. Perhaps you do

not recognise the symptoms which I have described as being significant of any disease which has been described in the text-books, but it belongs to a definite category of disorder known as Schönlein's disease. It is described under the name of erythema multiforme exudativum, or erythema polymorphicum. Either of these is a good name, because it indicates one of the characters of the eruption, namely, its variability in character. The eruption in this man has not always been the same; he has had different sorts of rash. He has had raised bumpy lesions, almost urticarial in quality, and erythema with hæmorrhage into it, sometimes called erythema hæmorrhagicum, the papular form, small slightly raised papules, and this simple purpura, which has extended into the legs and thighs, and occurred sometimes on the abdomen. The common lesions of purpura are not raised, and are indelible when you press upon them. In these cases the symptoms are very various and anomalous. They are generally seen in young adults, chiefly between the ages of twenty and thirty. They commonly begin with sore throat, as this case did, and there are pains in the joints, rheumatoid in character or truly rheumatic, swellings and tenderness in various joints, and very often there is gastro-intestinal disturbance. In this patient that particular element in the affection was well marked. But I have not told you of a more serious symptom. We have mentioned the copious vomiting, but in addition he had at one time very severe purging. He passed a good deal of blood and mucus in the stools, which were unequivocal evidences of a state of enteritis. So this man had an inflammatory state of the stomach and intestines. He thought the pain left his knees and other joints and receded into the stomach. From being articular, it became abarticular and visceral. That is what happens in some of these cases. Sometimes albumin, or even blood, appears in the urine. In this case, however, there was neither albumin nor blood in the urine. He is wasted and emaciated, and is distinctly out of health and in a cachectic state. Altogether, I think perhaps this is one of the worst cases of erythema multiforme that I have seen. You would not expect a man to be so long ill with this affection, and to be so resistant to the various treatments which we have adopted for him in these last three months. Though he is far from well, yet he is much better. At one time,

about five or six weeks ago, I hardly thought he would recover. I wish you to understand that erythema multiforme is not a mere skin disease. You recognise the skin lesions, but they should only serve to put you upon the track of the causal condition. They are symptomatic of some general morbid state, just as pleurisy is symptomatic of some other condition, constitutional and general. What constitutional state is this likely to arise from? I do not think you have far to seek for that. It is clear that in the majority of cases in which these various skin eruptions come out, whether they be raised or hæmorrhagic or erythematous, they are due to the poison of rheumatism. That is the peccant material which is probably at the bottom of the whole business in the majority of cases. In this patient you have not far to seek for evidences of that. You know that in many cases of rheumatic fever one of the first symptoms is sore throat; that is to say, in such case the rheumatic poison falls early and chiefly upon the fauces, and many a person who is going to have rheumatic fever begins by having sore throat. Within about a week of that occurrence pains in the joints come on, and then the other symptoms follow. This man had sore throat and then the articular symptoms, with effusion in the knees. Then there was the recedence of this process to the alimentary tract, with copious bilious vomiting and bloody purging, and then the recurrence from time to time of more or less stiffness in the joints and rheumatic pains. There was the persistence of the hæmorrhagic tendency, because these small patches of purpura mean hæmorrhages. I certainly think we cannot have a better explanation of this case than that it is due to rheumatic poison. Feeling sure that the whole of the symptoms he manifested were due to some toxæmia, we had the blood examined, and Dr. Drysdale made a careful examination of the blood, looking for the presence of any vicious intrusive microbes; but his research in that direction was negative. Still, his inability to find these must not be considered as proof that such toxic irritants were not, or had not been, there. Knowing, as we do, that rheumatic fever is an infective disease, due to a distinct microbe, which has been discovered by Drs. Poynton and Paine, so in this case we know we have to deal with an irregular manifestation of that poison. Erythema has long been recognised

as a rheumatic disorder. The erythema nodosum of young women is not at all uncommon, and it is well recognised as a rheumatic disorder, and very many erythematous rashes are distinctly due to rheumatic influence. The difficulty is to understand these abarticular symptoms, such as the vomiting and the passage of blood in cases of this kind; but it is not at all extraordinary, because our views hitherto on rheumatism have been too narrow; we have rather taken up the common idea of the layman, that anything which is painful, especially about the joints, is rheumatic; indeed, anything which is spoken of as "rheumatic" should now be taken to mean simply a pain. But the modern and more accurate conception of rheumatism is wider and larger than that, because we have learnt that rheumatism may fall upon different parts of the body, that there may be not only articular rheumatism but rheumatism affecting serous membranes; that the poison may fall most markedly upon the myocardium and produce myocarditis; that it may fall heavily upon the pericardium and cause pericarditis; or that it may chiefly show itself in the throat, and thus produce rheumatic tonsillitis; in other cases it may manifest itself on the skin and cause various erythemata; or it may fall upon sheaths, tendons, and periosteum, or on ridges of bone, and give rise to the well-known nodules. And I want you to bear in mind that these are all rheumatisms, and you will see that it is very different from the ordinary conception of rheumatism, namely, pain in a joint causing it to swell. And there is yet another rheumatism, that of the brain, which manifests itself in St. Vitus's dance or chorea. In connection with the disease we are considering you will find in the text-books of fifty years ago that the term applied was peliosis rheumatica; peliosis simply means purpura. This will show you that for a long time a form of purpura has been associated with rheumatism. That relation is established by the presence of joint-pain at some time or other before the occurrence of the hæmorrhagic petechiæ, so we really only widen our conception of the mischief which the rheumatic poison is capable of doing. Some people suffer more in their joints than do others. The rheumatic poison falls chiefly upon the weakest parts. In the case of a nervous child it falls upon the nervous system, of which the brain is the

centre, and you then have chorea. In a person with a weak throat it becomes rheumatic tonsillitis. Here, then, you have personal tendencies coming in to illustrate how diseases are borne and manifested. If the cause of this man's trouble is not rheumatic it is toxic of some other form; it is certainly due to some toxæmia. Therefore, our efforts in treating such a patient have to be directed towards the elimination of the poisonous products. Nature was evidently trying to drive out the poison by the stomach and bowels when he had the purgation and vomiting, and also partly by the skin. We have tried to do this still further by giving him full doses of calomel, and by medicines such as bicarbonate of soda and hydrocyanic acid, to check his vomiting. It was an old observation of Dr. Jeaffreson's, who thirty years ago was a physician to this hospital, that many cases of ordinary purpura do not get well until the patient has had rather smart doses of calomel. By the routine method in those days of giving three or four grains of calomel the liver seemed to be stimulated materially, and much good was done for the patient's general health. Just in the same way many cases of nettle-rash are found not to yield to the ordinary remedies, such as magnesia, until the patient has had a powerful emetic, which appears to clear out the alimentary canal and get rid of a good deal of irritating matter from the mucous surfaces. The purpura continuing and the pains having gone, we tried turpentine, which is a remedy in cases of this kind, but without any marked effect. We had to feed him carefully, on general principles, because of his weak stomach and constant tendency to vomit, and gradually he began to mend and to put on flesh. He still remained very ill and thin, although there was no pyrexia to cause wasting. He still needs great care, and as long as this purpura lasts there must be some mischief in his system which we cannot very well get at and localise. We have thought of the various sources of toxæmia, and amongst others that of a foul state of the mouth. Our attention has been a good deal directed to foul states of the mouth by the observations of Dr. William Hunter, especially in connection with pernicious anæmia; there are poisons generated by carious teeth and by a bad state of the gums. People thus affected are constantly swallowing poisonous matters and producing a kind of auto-

intoxication. We had our patient's teeth set in order, and provided him with a tooth brush and antiseptic powder, which, indeed, are necessary things for everybody. But neither did those measures seem to have any marked effect in this case. In the meantime we have to try to maintain his general health and increase it by proper food, in the hope that this hæmorrhagic tendency will wear itself out. This case becomes a little akin to a class of diseases with which it has been sometimes confounded, namely, infective endocarditis. This is a condition which should always be thought of when systemic toxæmia is present. Wherever you have erythema multiforme you cannot be sure that you have not a case of slowly infective endocarditis in which the patient is becoming poisoned by infective particles carried off his diseased valves, giving rise, indeed, to an arterial pyæmia. This man had no sign of heart disease; the rheumatism has not affected his endocardium. These cases do simulate infective endocarditis because you also meet with skin lesions in infective endocarditis, embolic conditions which superficially resemble those of erythema multiforme.

I thought it would be well to bring before you an example of a disease which you may meet with, and I have brought you one of the worst cases of the condition that I have seen myself. As a rule, you may consider that the disease will pass off in about six weeks or two months. This has lasted three months. Probably the bad weather we have had during the last three months, with its constant changes, may have particularly encouraged the rheumatic germ, as it probably has the influenza germ. And then you have also to consider the effect of environment. If we had moved him away from the neighbourhood of Smithfield and could have had him transferred to some fresher air he might have got well much quicker. To put a patient to bed does not necessarily mean that he will get well; it may be necessary to move him to a better climate. In chronic cases of toxæmia you may find the patient will go on from week to week and month to month without marked improvement, but if you convey him to some other place and give him a complete change of environment he may not seldom get rapidly well.

A CLINICAL LECTURE
ON
THE SURGICAL TREATMENT OF
INFANTILE PARALYSIS.

Delivered at the Hospital for Sick Children, Great Ormond
Street, W.C.

By F. J. STEWARD, M.S., F.R.C.S.

BEFORE I go into the treatment of these cases of infantile paralysis I think it is well worth our while to consider a few points regarding the pathology of the disease, because they have such a very important bearing, as you will see later, on the question of treatment.

First of all we will consider the answer to this question, which I will write up on the board: *What may happen to a muscle that is paralysed as the result of anterior poliomyelitis?* If you think for a moment you will see that there are three possibilities in connection with that question.

The first is this: that the muscle may completely recover. Of course, when an attack of infantile paralysis occurs, the usual thing is that a number of muscles are completely paralysed at the time. After a little while these commence to recover, and at the end of six months to a year you will find that a good many of these muscles have completely regained their power. We know, then, that muscles may completely recover their power, though it may be a long time after the attack.

The second possibility is that the muscle may partially recover, that is to say, some of the muscular fibres belonging to the paralysed muscle remain paralysed, and eventually atrophy, and are replaced by fibrous tissue, while the rest of them recover their power. Now there is a very important result of that partial recovery, and it is this: that the natural tone of the muscle is diminished, in consequence of which the muscle tends to be stretched by the action of its opponents. In other words, we may say that if a muscle partially recovers from an attack of infantile paralysis that muscle is liable to become unduly stretched.

There is a third possibility, and that is that the muscle remains completely paralysed, and in that case the whole of its muscular fibres are replaced by fibrous and fatty tissue, and in addition the same results follow as in the muscle which only

partially recovers, namely, that as it has no resiliency or tone of its own it is unduly stretched by the action of its opponents. Let us take an example, we will say of a case of paralysis of the extensors of the foot. The consequence of this is that the paralysed and relaxed muscles become stretched and lengthened by the action of the muscles behind, and the heel is raised; in other words, talipes equinus is produced. Now we have answered our first question, and I would like you to bear in mind the three possibilities that we have considered.

There is another important question from the pathological point of view that we might consider with advantage for a moment, and it is this: *What results may partial or complete paralysis of a muscle or group of muscles produce upon the unparalysed opponents of that muscle or group of muscles?* That is a very important question, and I think it is well to consider the answer to it at different periods. In the early stages the effect on the opponents is comparatively small; in fact, the only thing noticeable is a tendency to over-contraction. This, of course, is owing to the want of opposition, and is at this stage obvious only when the whole of the muscles of the limb are put into action. Then, as time goes on and the paralysed muscles begin to stretch, a shortening of the non-paralysed group of muscles takes place, and the important thing to remember is, that in the first place, the shortening is a *physiological* one only; in other words, it is simply an increase of tone. To give you an instance. Suppose we are dealing with a case of paralysis of the anterior tibial muscles and the extensors of the foot; gradually, as time goes on, the tendo Achillis shortens so that the position of equinus is produced. Now, in the first place, this is *physiological shortening* or *contraction*, as you can see by grasping the foot and making an attempt to replace it in the normal position. That you can do perfectly easily, because you can overcome the physiological contraction in the gastrocnemius and soleus. Later still, however, further changes take place, and the physiological shortening or contraction of the opposing muscle is replaced by a *pathological* shortening, a structural change in the muscle associated with fibrosis, which is known as *contracture*. The result is that in this condition, to keep to our old example, that of talipes equinus,

due to shortening of the gastrocnemius and soleus, and therefore of the tendo Achillis, you will now find that you cannot overcome the deformity and replace the foot in a normal position, owing to the fact that a true shortening or contracture is present, and not a physiological one or contraction. Thus, there is permanent alteration in the position of the foot, which has got into the position in this case of talipes equinus. That, again, is a very important point as regards the treatment, as you will see presently.

In addition to the two changes I have spoken of in the muscles there are other changes which occur in the later stages of infantile paralysis and which affect other tissues, namely, the fascia, the ligaments, and later still, the bones themselves, and these changes, in fact, constitute a tendency to render permanent the contracture which is produced by the shortening of the opposing muscles. For instance, the condition of pes cavus, which results after talipes calcaneus. Supposing that the gastrocnemius and soleus are paralysed, the effect is that the heel drops and that the structures between the two pillars of the arch of the foot contract and draw these two arches towards one another. One of the pillars is formed, of course, by the toes, roughly speaking, and the other by the heel. Shortening of the plantar fascia and the muscles and ligaments in the sole takes place, with the result that talipes cavus or pes cavus is produced as a direct result, you will see, of the paralysis of the gastrocnemius and soleus, and eventually, if years are allowed to pass, you will find that actual bony changes have taken place which render the position of pes cavus a permanent one.

Now from what I have just been over with you as regards the pathology you will see that the conditions in which a limb may be left after an attack of infantile paralysis may vary immensely. This is, of course, owing to the possible variations in the different factors which enter into any particular case, these chief factors being the particular muscle or group of muscles paralysed, the degree of paralysis of each, and the changes which take place in the opposing muscles. All these factors in a case may vary, and consequently the resultant conditions, as you will see by the cases I am going to show you, are very numerous. But speaking generally, apart from exceptional cases, which I will leave out of consideration, I think it is possible

to divide cases of infantile paralysis into three distinct classes, which I will illustrate to you directly when we have the patients in, and I think this grouping is useful from the point of view of treatment in particular.

The three classes are these :—(1) Those cases in which the amount of paralysis is comparatively small, affecting usually either one muscle or the muscles of one group, or sometimes of two groups, the other muscles being comparatively healthy.

It is certainly the largest class, and all the cases that I am going to show you, with the exception of one, I think, belong to it. As an instance, there is the common one of simple equinus, in which, from weakness, that is, partial or complete paralysis of the anterior muscles, the gastrocnemius and soleus contract, and, again, there is the condition of calcaneus, when the opposite takes place. At any rate, you will find that this group is the largest group, and also that it is the most amenable to treatment.

The next class consists of those cases in which the recovery from the attack of paralysis is less complete. There is general weakness owing to failure of recovery affecting all or the majority of the muscles, some of the muscles, or groups of muscles, however, recovering to a greater extent than the others. Now this is not a very uncommon group. You will find that as a result of the partial paralysis of practically all the muscles, there is a general weakness of the whole limb, but owing to the fact that some muscles have recovered their power to rather a greater extent than others, there is a tendency to the production of deformity at the same time. For instance, a common condition is that in which gastrocnemius and soleus recover their power better than others, with the result that there is a certain amount of contraction or shortening of the tendo Achillis, and the condition of talipes equinus results. Another common result is to find that the muscles are all weak, but that the peronei have recovered their power better than the others, and in that case they tend to displace the foot outwards, and to produce the condition of talipes valgus.

We now come to the third class of cases, and that includes those cases where there is complete permanent paralysis of all the muscles. I am speaking of the leg of course. The result is a flail-like condition of the ankle and foot generally,

and associated with that there is general coldness and blueness of the skin, and a tendency to chilblains and other trophic changes. Trophic changes also take place in the joints, and in addition there are important changes in the bone, so that an atrophic condition of the bone appears very similar to that seen in old age, resulting in a tendency to fracture, and moreover the loss of nerve influence and blood-supply to the bone result in a slowing down of the processes of growth, so that usually the paralysed limb will be very much shorter than the other one.

Now I think I had better have the cases in and show you them one at a time, and then we can go into the question of treatment.

CASE 1.—This is a patient *æt.* 3 years, who, I think you will see, falls into practically the third class. The whole of this limb is extremely wasted, the foot and leg are cold, and you will notice there is a flail-like condition of the ankle and foot. There is very little power over the foot and ankle at all. He has a little power in the leg, and it is in the *tibialis anticus*. Sometimes you can see a slight contraction in the *tibialis anticus*, but this case, for all practical purposes, falls into the third group of the cases I have mentioned with almost complete wasting. All the muscles are flabby and quite soft; there is no feeling of tone about them. In addition to that, there is marked shortening of the limb. He suffered from an attack of paralysis eighteen months ago, and both lower limbs were completely paralysed at the time. The other limb, the left, has completely recovered.

CASE 2.—This is a little girl *æt.* 4 years, who had an acute attack at the age of about twelve months, and here there is much better recovery than in the case just shown you. If you will compare the two legs you will see that there is not such a very great difference in size of the upper parts, but lower down you can see that the left leg is a good deal wasted on the inner side and also at the back, and there is a peculiar condition here that you do not often see, and it is that prominence in the upper part of the back of the leg which, I take it, is due to the recovery of the two heads of the *gastrocnemius* without recovery of the *soleus*. There is also slight contraction of the *tendo Achillis*, pointing to weakening of the extensors, and when she moves her foot you will see that the position of valgus is produced. I think everybody

can see the valgus position of the foot that is produced by the contraction, and you can see, moreover, quite distinctly that the *peroneus tertius* is contracting, and the *peroneus brevis* also. Both of these are acting well. There is, therefore, paralysis of the *tibialis anticus* and probably of the *tibialis posticus* as well, and to some extent of the extensors. As a result there is a certain tendency to equinus, and distinct tendency to valgus, so that it is a case to be described as *talipes equino valgus*, and it falls into the first group.

CASE 3.—The third case is a little girl *æt.* 2 years and 8 months, who forms an interesting contrast with Case 2. You will see that the foot is in the condition of *calcaneo-valgus*. There is partial paralysis of the *gastrocnemius* and *soleus* resulting in stretching of the *tendo Achillis*, together with distinct paralysis of the *tibialis anticus*. On the other hand, the *peronei* act well. In addition to that, in this case, there is a good deal of coldness of the limb, and in that respect it rather more approximates to the first case I showed you, for there is a tendency to the flail-like condition of the foot, although there is better recovery here than in the first case. One might sandwich this case in between the first and the second cases, where there was good recovery, except in one or two muscles. So that here the *tibialis anticus* and *posticus* are the worst, and there is weakness of the other muscles, chiefly the *tendo Achillis*, to put it shortly, though you will remember, of course, that the *tendo Achillis* is not a muscle.

Now we come to Case 4. This little girl is *æt.* 3 years, and the attack commenced eighteen months ago. Here, again, the common result followed, namely, paralysis of the *tibiales* muscles, and you will see that there is a tendency to valgus. On her moving the foot as she is sitting, there is some increase of the valgus, but perhaps on the whole, it is not very much. Now, in addition to that, you will see that there is slight weakness of the *gastrocnemius* and *soleus*, allowing a greater flexion than normal to take place, so that there is a little tendency towards *calcaneus*, and, moreover, you can see here very well a thing which is sometimes noticeable in *talipes calcaneus*, and that is the peculiar globular shape of the heel, for it forms much more of a rounded eminence than it normally does. There is another point which I can show you in this case, and that is how to ascertain

whether the contraction present is simply a physiological shortening of the opposing muscles or a contracture. Now, the active muscles producing the deformity here are the peronei, and, you see, they are shortened, that is to say, that the foot is held in the valgus position. On taking hold I can produce the varus position quite easily, so that there is clearly simply a physiological shortening, and not a contracture. Here, then, the tibial muscles are chiefly affected and the tendo Achillis slightly.

CASE 5.—Here is another patient. He is a boy *æt.* 6 years who has had paralysis for five years. Looking at the leg you can see there is a good deal of wasting, and the natural position the foot is held in is one of varus and slightly of equinus. When he moves the foot to put it down you can see the tibialis anticus contract quite well, and you can feel the tendo Achillis quite well, but he has lost power in the peronei. Now we must apply the test to see whether the tibial muscles are really shortened. You see they are to some extent, for the foot will not go quite out to the valgus position, but it will very nearly. There is then real slight shortening of the tibial muscles on the inner side, and a good deal of loss of power in the extensors, so that it is in just the opposite condition of the case we have just had in.

We will note then Case 5 is equino varus. Paralysis affects the extensors and the peronei.

CASE 6.—The sixth is the last one. Here is a boy who is in the ward at the present time. He is twelve years of age. There is a good deal of shortening of the limb, probably more than in any of the other cases. The history of the attack is that it occurred at the age of four years. There is a considerable difference in the two limbs, and there is a certain amount of general wasting of this leg, so that in the first place you would put the case into Group 2. All the muscles are partially paralysed, but again some of them are affected more than others, and you will see here that the foot has dropped a bit, and there is a slight increase in the concavity. Now let us see him move it. Here, again, when the patient moves the foot the position of valgus is produced. His electrical reactions have been tested, and the result is this: that the tibialis anticus does not react either to the galvanic current or the faradic current, the tibialis posticus reacts only slightly, the peronei

quite well, the extensors are weak, and the gastrocnemius and soleus are strong; so that the general result is that he is weak on the tibial side, and also in the front muscles. The result is a contraction of the tendo Achillis which has produced this condition of equinus. The foot will not go up to the position of a right angle. Further, that contraction is a permanent one, namely, a contracture. There is also a tendency to valgus from the over-contraction of the peronei.

Now we must go into the question of treatment without further delay.

I will take the three classes one at a time, and it is quite clear in the first place that the treatment of cases which fall into Group 1, namely, where there are only a few muscles paralysed, and usually the muscles belonging to the same group, is the most satisfactory. Until quite recent years the treatment of these cases was founded practically on wrong principles. Supposing we take one or two of these cases we have seen together to-day as examples; here is a case of talipes equinus due to paralysis of the extensor muscles. There is a contraction of the tendo Achillis and a certain amount of contraction in the peronei. All that would have been done a few years ago would have been to diminish the power in the tendo Achillis and to lengthen it by division. Tenotomy results in diminution in the power of the muscle, and also in lengthening, and in that way the deformity may be overcome. But you do not give the child any increase of control over the foot, and the probability is that he will have to wear an apparatus, which is not only expensive but heavy and cumbersome, for the rest of his life. Another treatment which used to be carried out a good deal was the treatment of talipes calcaneus by shortening the tendo Achillis. The tendo Achillis had been lengthened as a result of paralysis of the gastrocnemius and soleus, and the effect of shortening it and cutting a bit out and splicing it is simply directed to overcome the deformity for the time. As soon as the child begins to get about again, relapse is almost sure to take place, because the opposing muscles still act too strongly and stretch the tendo Achillis again.

Now let us consider for a moment what the indications for treatment really are in a case like this. Let us suppose that A B represents the foot with a muscle attached to it on either side. We

will suppose now that the muscle C is paralysed. What is the result? Why, the muscle C will be stretched and lengthened by the increase in tone, and therefore shortening of the unparalysed muscle D, and the foot is displaced. One muscle has all the power, and the other has none of the power. The obvious thing to do if possible is to transfer some of the power from B to A, and so bring about a proper distribution of power again, or, at any rate, to increase the power on the weak side, so as to help the weak side to balance the strong side. In addition to that it will be necessary to overcome the deformity by doing away with the shortening of the muscle D on the unparalysed side, and with the lengthening on the weak or paralysed side. Well, now, all this can be done by the treatment which has been only comparatively recently introduced, and that is the transplantation of tendons or muscles; in other words, the transplanting of healthy muscle, or healthy tendon attached to a piece of healthy muscle, on to the paralysed one. The length of the shortened tendon can, at the same time, be increased by tenotomy if this shortening is due to a contracture.

Now let us go into this question of tendon transplantation a little more carefully. There are certain important points to consider. The first thing is this: that in order to produce a proper condition of the foot and to prevent a relapse into the previous deformity, the indication is to produce such a distribution of the power that remains that the muscles of the various different groups will be balanced one with the other. Secondly, the next indication is to do away with the deformity so that the new position in which you put the foot shall be the correct one. Other points, which are extremely important, I will just mention to you. The first is, that the operation must be carried out under aseptic principles. If any supuration takes place in the wound (for there is generally a large one owing to the exposure of the muscles and tendons operated on) severe adhesions may take place, and so the limb may be further crippled. Moreover, the stitches which you put in are bound to work out.

The next point which arises is this: Which of the muscles is to be chosen for the transplantation? There are two distinct ways of going about it. One is to transplant on to the paralysed tendon the *nearest* healthy muscle. The tendon which is

nearest, of course, in the first place, will need less disturbance, since it is also the nearest in function, the new connection will be all the more quickly learnt by the patient. The other plan that can be adopted is to transplant one of the active opposing muscles that are at a distance. To give you an instance, supposing the tibialis anticus is paralysed, and that the peronei are in a good condition, according to the first plan you would transplant the nearest tendon, which would be the extensor hallucis proprius, on to the tibialis anticus; but in accordance with the second suggestion you would transplant the unparalysed peroneus brevis on to the paralysed tibialis anticus, so that you have two good muscles, one on the inner side and one on the outer side, and, as a result, there would be a balance of power between the two. Some operators have preferred one method and others the other. There is a case I operated upon in this hospital with a satisfactory result, so far as I know at the present moment. The operation was done not so very long ago—about four months ago, or perhaps a little more. The patient was a boy *æt.* 6 years, with talipes valgus, due to the paralysis of the tibialis anticus. The peronei acted well and so caused the valgus. So I transplanted the peroneus brevis on to the paralysed tibialis anticus. The foot is now in a perfectly good position, and can be moved about in all directions. The valgus has completely disappeared, owing to the fact that one of the peronei muscles was transplanted to the inner side.

Another point is very important, namely, to correct any other deformity which may be present before you do a transplantation operation. For instance, supposing there is contraction of the plantar fascia, we must divide that fascia and get the foot in a good position. Then, again, if certain tendons are permanently shortened from contracture, it is important to overcome that permanent shortening, either by lengthening the tendon or by subcutaneous division of the tendon, and allowing it to unite afterwards. The next and fourth point is in every case to overcome the deformity due to the lengthening of the paralysed tendons, by fixing the transplanted tendon so as to support the foot in the corrected position. To give you an instance of what I mean, supposing the toes are dropped and there is a condition of valgus, and it has been decided to transplant the peroneus brevis on to

the tibialis anticus, do not fasten the two muscles together so that they are in a relaxed condition, but fasten them together so that the foot is held in the corrected position; then you will have overcome the lengthening which has taken place in the tibialis anticus.

I need not go into the minute details of the operation, but will illustrate it by describing one of the cases upon which I have recently operated.

This was a boy *æt.* 6 years, who had pes cavus and talipes calcaneus. The heel was dropped and rounded and ball-like, and there was considerable contraction of the structures in the sole. The peronei acted strongly, but the gastrocnemius and soleus were extremely weak, so that the tendo Achillis was lengthened. The first thing that I did was to subcutaneously divide the plantar fascia and some of the ligaments and muscles in the sole, and get the foot straight. The next thing was to make an incision about four inches long on the outer side of the tendo Achillis, and dissect out the tendo Achillis and the peroneus longus. Then I transplanted the peroneus longus tendon on to the outer side of the tendo Achillis, fixing it there with silk sutures. I used about six, and in order to make the junction as firm as possible, I split the tendo Achillis and passed the tendon of the peroneus longus through it, and stitched it both to the front and back of the tendo Achillis. The wound was then sewn up, and the leg put up in the correct position in plaster of Paris. At the end of five weeks I took off the plaster, and I found the foot in a good position, but more than that I cannot say as to the result at present, as sufficient time has not elapsed. The lengthening of the tendo Achillis has been overcome by shortening it up in that way, and the tendency to valgus by getting rid of the peroneus longus and making it into a new gastrocnemius. Thus are roughly sketched the stages of the operation. But of course they will vary, as I have said before, with different cases, since the cases themselves vary so as regards the muscles affected.

Now suppose we consider some of the cases I have shown you to-day, and the treatment applicable to them. No. 2 was a case of equino valgus. Here the paralysis affected chiefly the tibialis anticus, and to some extent the posticus and the extensor longus digitorum. What muscle can we transplant so as to make a new anticus? The

nearest tendon is the extensor longus hallucis. The next one on the outer side is weak and not good for transplantation, and after this we come to the peronei. These are overacting and producing the condition of valgus, so that if we take some power away from the outer side and put it on the inner side by attaching the peroneus brevis to the tibialis anticus, we may get a balance of power; and that is what I propose to do in this case.

In Case 3 we have a condition of calcaneo valgus. There, again, the anticus is the worst, but there is a good deal of general weakness affecting, you will remember, the tendo Achillis. This case really falls into the second group of cases, and *à propos* of the treatment of the second group of cases, one has to be very careful as to what is done. They cannot, owing to the general loss of power, be expected to do so well after tendon transplantation as the other cases; but if there is one muscle in good condition, or one group in good condition, it is quite conceivable that one will be able to overcome the deformity. Here, however, there is no particular muscle which stands out as being in extremely good condition, and in that case I think it will be quite useless to attempt anything in the way of operation. The only thing to do in cases like this is to provide the child with an artificial muscle to overcome this weakness of the tibialis anticus, and possibly he will want some other apparatus, but that is all you can do for the second group of these cases.

In No. 4 we have another case of calcaneo valgus, but with very much less paralysis. It affects the tibialis anticus and posticus chiefly and slightly the tendo Achillis. Supposing in this case the peroneus longus is transplanted on to the tendo Achillis, as I did in the case I have just described to you, two things are accomplished. In the first place, the weakened tendo Achillis is strengthened, and it is shortened at the same time by pulling it up so as to overcome the tendency to calcaneus. At the same time, by taking away the peroneus longus from the outside and making it into a posterior muscle you do away with some of the strength of the peronei, and thus overcome the tendency to valgus, so that what is clearly the correct treatment in a case like this is to transplant the peroneus longus on to the tendo Achillis, and that is all I intend to do.

In the fifth case, which is one of equino varus

(just the opposite condition to the one we have been considering), the weakness affects the extensor muscles of the foot and also the peronei. The tibialis anticus and posticus are, on the other hand, in good condition, and we want to get a new peroneus muscle and a new extensor if possible. But the extensors are not nearly so important as the peronei. If you will just think of the possibilities anatomically you can see at once that the obvious thing to do is to split a piece off the tendo Achillis and transplant that on to the peroneus longus, by which means a new peroneus longus is made and the tendo Achillis is weakened, and so some of the tendency to equinus is overcome. You will remember also that in this case some of the equinus was permanent. It is as well to get rid of that at the same time, and this is easily done by dividing the rest of the tendo Achillis; and in that way I hope a satisfactory result will be attained.

Time will not allow me to go fully into the question of treatment of the other cases, that is to say, the third group, where there is complete paralysis and shortening of the limb and so on, but in many of these cases a fairly satisfactory result can be obtained by stiffening the ankle-joint, and in a few cases it is necessary to stiffen the knee as well; in other words, to perform the operation of arthrodesis. The interior of the joint is exposed and the cartilage shaved away, so as to produce a stiff joint. In some of these severe cases, however, amputation gives the best result.

MESSRS. BURROUGHS, WELLCOME AND Co. have recently introduced "Soloid" Sodium Hydroxide and "Soloid" Pyrogallie Acid to provide a ready means of estimating the degree of oxygenation of a sewage effluent. The examination by means of the above "Soloid" products is simple. Fill a wide-mouthed bottle, holding about 4 ounces, with the effluent as it runs from the beds, drop in one "Soloid" product of each reagent, and insert the stopper carefully, and in such a way as not to include any bubble of air. Shake the bottle for about half a minute, and note the effect produced. If the liquid remain colourless or only faintly coloured, the oxygenation is not satisfactory; if it assume a rich red-brown colour the oxygenation is satisfactory.

THE PRESENT AND FUTURE OF ELECTRO-THERAPEUTICS.*

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GENTLEMEN,—It gives me very great pleasure to be addressing an electro-therapeutic society in this country. Those who practise medical electricity, few and far between as they are, are apt to feel a certain loneliness at times, because they so seldom have the opportunity of meeting congenial minds with whom to discuss the various points of practice which are interesting them at the moment. In England we have been particularly badly off in this respect, but I hope that the foundation of this Society marks the beginning of a new era.

Three years ago I attended the Congress of the French Association for the Advancement of Science at Boulogne. There we had daily gatherings of twenty or thirty members, all keenly interested in electro-therapeutics, and excellent papers were read. There is no doubt that the interchange of ideas is stimulating and refreshing; I have felt from time to time that I had some idea which I should very much like to talk over with those who were competent to advise upon it and to discuss it with me, and sometimes, merely through lack of such opportunities, the idea has remained a dormant one.

It is now twelve years since I began systematic work in medical electricity, and owing to the rapid progress which electricity has made since then, it is easy to recognise that great changes have occurred.

It will be interesting to consider for a moment how medical electricity stood then, and to compare its position with that which it holds now. At that date the treatment of uterine fibroids by electricity, after Apostoli's method, was on trial. The treatment of urethral stricture by electrolysis was in the same stage. Dr. Steavenson, my predecessor at St. Bartholomew's, had just published some admirable papers on the electric bath. To-day, the electrical treatment of fibroids and of urethral strictures has practically disappeared. The electric bath, on the other hand, has established its claims to recognition, and now holds a stronger position

* Delivered before the British Electro-Therapeutic Society on February 14th, 1902.

than it did then. It is useful to consider for a moment why this is so.

Electric methods of treatment to succeed must not only be able to achieve a successful result, they must do it more easily or more certainly than the other competing methods. Merely to do as well does not compensate for the introduction of the electrical apparatus, and the time and trouble in learning how to handle it. Apostoli's method failed, not because it was useless, but because it was unable to compete with other established procedures. The same is true of the electrolysis of stricture of the urethra.

Of the things which were firmly established at that date, and still hold their ground, I may mention electrical testing. The principles of electrical testing by induction coil and battery current, as built up on a firm basis by Erb, have held their ground indeed, but have not made very much progress since his time, not so much because testing as we have it to-day is perfect by any means, but because of the difficulties in overcoming certain of the obstacles involved, of which the chief, and perhaps the most interesting, is the measurement of induction coil currents or "faradic" currents. In a paper which I read before the Institution of Electrical Engineers two years ago, I hinted at the possibility of replacing the induction coil by a condenser, in order that the discharges employed for stimulating the muscle should be discharged at a measured voltage from a condenser of measurable capacity. In that way, I ventured to say, a method of getting round the difficulty would be found. Something of the kind had already been proposed by a Dutch investigator, Dr. J. L. Hoorweg,* but much remains to be done. I have myself at the moment some experiments in hand for utilising condenser discharges for electrical testing, which are based upon the method known in connection with the statical machine as Morton's method.

Other small points to be noted in connection with electrical testing are the "longitudinal reaction," by which is meant the setting up of muscular contraction in a muscle by stimulating its distal end by a current which traverses its fibres longitudinally, and the clearing up of that

complication in testing which takes note of the relative strength of the anodal and cathodal closing contractions in health and in disease. This "polar formula," as it is called, with its reversal in cases of the reaction of degeneration, is certainly not a constant phenomenon in all stages of that condition, so far, at least, as my experience goes. Another matter which now has to be considered in all comparisons of electrical testings is the importance of the self-induction of the testing circuit in modifying the results obtained. Dubois has clearly proved that the current necessary for the minimal contraction requires to be considerably increased when the testing circuit includes a portion having a high self-induction, and he attributes this to the fact, which has long been well known, that self-induction in a circuit tends to retard the rate of growth of current in the circuit at the moment of closure. Thus the presence of a galvanometer in a circuit has this effect by diminishing the suddenness of the shock to the muscles. Electrical testing is not completely satisfactory because it does not enable us to distinguish between the reactions which accompany a partly damaged nerve and those which signify a severed one, and it cannot yet give clear indications of commencing recovery in a recently sutured nerve. An answer to these questions, so often asked, cannot at present be supplied directly by an electrical test. That is its chief weakness.

A small point to which I would wish to draw attention, as one which deserves further examination, is the remarkable persistence of the reaction of degeneration in certain cases. For example, in infantile paralysis I have on several occasions noted the presence of a typical reaction of degeneration ten or even twelve years after the onset of the attack, although in cases of division of nerve-trunk left unsutured the reaction of degeneration fades away in a much shorter time. This suggests the somewhat paradoxical thought that the persistence of the reaction of degeneration in a muscle implies something short of complete detachment of the muscle from its nervous centres, and at present no explanation can be suggested.

A prominent feature which reveals itself in a review of the past twelve years is the development of public supplies of electricity by electric lighting mains, for this has probably done more than any

* 'Recherche sur l'Excitation électrique des Nerfs,' Haarlem, 1899.

other thing to help medical electricity. The electric lighting mains have not only given us a source of current without the trouble and expense of batteries, but through them we are able to employ magnitudes of current and high pressures which were not to be thought of a few years ago. The mains have also given us a new type of current, the sinusoidal current, which has been adopted for therapeutic purposes by reason of its great advantages in quite a large class of cases.

Of late years the statical machine has also been taken up anew, and probably it has not yet reached the maximum of its employment. To me it seems still to have latent possibilities that may yet be brought out by patient investigators. The presidential address of Dr. Macintyre to the British Laryngological, Rhinological, and Otological Association is full of significant matter connected with the static machine, inasmuch as he there records successful cases in the treatment by static methods of lupus, of rodent ulcer, and of carcinoma. At St. Bartholomew's Hospital we have several cases of lupus at the present time under treatment with the static breeze, and the results appear to be most promising.

Another prominent feature of the electro-therapeutics of the day is the high-frequency apparatus. At the present time it is passing through what might almost be called a boom, and various valuable therapeutic qualities are attributed to it. Reduced to its simplest form, the basis of the claims made for this apparatus may be stated somewhat as follows:—D'Arsonval, by scientific experiment, succeeded in proving that high-frequency applications produced an output of heat, an increased expiration of CO_2 , and an increase in the urea, both in animals and in human beings. In a word, he established the fact that the *catabolic* processes of the body can be considerably augmented in this way. That we may accept, I think, without question; but when the inference is drawn for us that the increase of *catabolic* processes implies the setting up of acting *anabolic* processes afterwards, we cannot help asking for more direct proofs. Experiments upon cases of diabetes with high-frequency currents have frequently been reported, but the results have been most disappointing, for, although a diminution in sugar during treatment has been established in a number of cases, yet that is not the

same thing as a cure of the diabetic condition, and for the most part the cases reported on remained diabetic afterwards. There is one general trophic effect which we must not forget in this connection, and that is an influence in the direction of reducing obesity. This effect may be regarded as much more catabolic than anabolic in nature, and therefore as being much closer to the observed physiological action of this treatment than many of the other things for which it has been proposed. In various skin affections, the local effect of the *effluve*, or brush discharge of the apparatus, has been found to be remarkably useful.

In the case of tubercle and tubercular diseases, numerous electrical methods have been suggested in the last few years, and, curiously enough, the methods have been of the most diverse sorts; for example, sparks from a statical machine to the chest, general electrification, the brush discharge of the statical machine, auto-conduction, and other high-frequency methods—all have afforded one or more successful cases. But it is too soon yet, in my opinion, to accept with conviction the existence of any real and constant good effect from electricity in these diseases. Different as the modes of treatment are, there is one common factor in them all, and that is the production of ozone, and it is not impossible that the ozone may play a part in the good results obtained.

A field which is much in need of cultivation is that of the treatment of skin diseases electrically. Numerous cases are on record which show that the action of electrical currents upon various forms of skin disease is decidedly useful, and the time is now ripe for a systematic application of electricity to skin diseases in order to determine its powers and its limitations. Obviously this work must be done by those who make a study of cutaneous disorders, and it may be laid down as an expansion of this particular statement into a general one that electrical treatment cannot be expected to advance rapidly until its uses for each group of morbid conditions are tested by those who are specially qualified in the class of disease which is to be studied. The electrical specialist cannot be expected to be also specialist in all the branches of medical work. His position is, in a sense, anomalous. He cannot be first-rate in all the lines of special work. Applications of electricity in diseases of the eyes or the ears, the nose or

the teeth or the chest, ought to be studied by specialists in those subjects.

Among the collateral branches of physical science, which promise just now to have a prominent effect upon the practical therapeutics of the future, are the effect of blue light, which has lately been stated by a Russian observer to have very marked anæsthetic action upon the skin, sufficient to permit of minor surgical operations being done without pain; the action of ultra-violet light, which is just now being tried by Dr. Hugh Walsham and myself in St. Bartholomew's in the treatment of lupus; and the Becquerel rays. The Becquerel rays are among the most astonishing things of the present time. They appear to occupy a position intermediate between the ultra-violet rays and the X rays, and are emitted by numerous chemical compounds, particularly by compounds of uranium, thorium, or barium. Doubtless you may already have heard of the peculiar effects, like those of X-ray burn, which were experienced by Professor Becquerel on his own person as a result of carrying in his pocket a small capsule containing a specimen of highly radio-active material. The radiations of the substance acting through several layers of clothing, and continued only during parts of two days, had the effect of producing a burn similar to the well-known X-ray burn, and corresponding in shape and in size to the capsule which he carried in his pocket. The speculations which have been indulged in as to the cause of X-ray burns have not yet resulted in any definite conclusion. We are still not able to settle whether the X-ray dermatitis is due to the X rays themselves, or whether it belongs to the electrostatic discharge which occurs around the X-ray tube, or to some other associated phenomenon.

In the case of lupus there are successful results to show, not only from treatment by X rays, but also from treatment with ultra-violet rays, from treatment with the brush discharges of the statical machine, and of the D'Arsonval apparatus, and from treatment with the blue light of Finsen's arc lamp. I say the blue light of Finsen's lamp, because I think it probable that the light given out by the Finsen apparatus is probably very poor in ultra-violet rays, because of the ease with which these rays are absorbed, not only by glass but also by rock crystal; and considering the various media

which are interposed between the source of the light in Finsen's apparatus and the surface of the patient's skin, I cannot help thinking that most, if not all, of the ultra-violet rays must have been filtered out before the radiations of the lamp arrive at their destination.

The treatment of cancer, of course, is the most attractive of all the possible applications of Röntgen rays and other radiations to therapeutics at the present moment. Most of us, no doubt, have cases in which we are making guarded trials in this direction. It is too soon yet to express any very definite opinion, but it is not too soon to relate any interesting cases which may come under our observation. At St. Bartholomew's we have been entrusted with the treatment of a case of late cancer of the breast, a case of recurrence *in situ* after operation. The patient when sent to us had a large open ulcerated wound of the right breast, measuring about four inches in one diameter and five in the other. Treatment with X rays was commenced on November 14th, and an exposure of ten minutes was given four times a week with a few omissions until January 17th. After a few days signs of healthy cicatrisation began to appear round the margins of the ulcer. This continued to advance until the healing process had extended inwards from the margin for a distance of fully half an inch all round. Curiously, the new epithelium was deeply pigmented, the centre of the wound gradually ceased to slough, and appeared to be in process of healing all over; but, unfortunately, the patient being at a late stage of her illness, secondary deposits in remote parts of the body became very disagreeably evident. The patient suffered a spontaneous fracture of both the femur and the humerus, and the X-ray treatment was suspended in view of the hopelessness of the future of the patient. The total number of exposures was seventeen, and it is very important to note that many were made with the ulcerated surface covered by dressing or bandage. Since the suspension of treatment, exactly four weeks ago, the process of healing has gone on quite steadily, and now there is a firm and healthy scar covering the whole surface, except about one square inch in the centre. This portion is also a healthy healing surface though not yet covered over by epithelium. Thus nineteen twentieths of the area of a cancerous ulcer has healed up in three months as a consequence of

X-ray applications, and the result is one which obviously is a most significant one. The local effect was splendid, but there was no influence upon the spread of the disease in the remote parts of the body. Indeed, it would have been unreasonable to expect it. We must hope for the opportunity of treating some cases of cancer in the early stage, when the disease is still limited to a superficial part, for when the disease is no longer local, but has implicated distant and deep-seated organs, it is too late to apply X-ray treatment to the original site of the disease. From observations up to date, rodent ulcer appears to be completely controlled by X-ray treatment. Quite a number of cases have been reported by many observers in different parts of this country, as well as abroad, of the successful treatment of rodent ulcer by X rays; and from its close relations with epithelioma, one may be allowed to hope that simple epitheliomata, such as of the lip, will also by-and-by be successfully treated by X rays.

These are the points upon which the greatest interest is concentrated at the moment in electro-therapeutic circles, but it will take time to show how far this mode of treatment is likely to be a permanent success.

A very serious matter is that of the encroachment of unqualified persons upon this field of work. For example, ten years ago there was a traffic in electric belts which had attained enormous dimensions, so that it was estimated that £70,000 was spent yearly in advertising them. After that the electric bath was dragged in the dirt in connection with massage shops of bad repute; since then there has been a boom in hot air contrivances, electrical and other. At present the high-frequency apparatus is in danger of becoming the stalking-horse of the quack. To-morrow it may be that the curing of cancer by the X rays may be the vogue. All these things are serious, and difficult to combat; the employment of the physical forces heat, light, and electricity for treatment implies the use of machines and instruments, and the instrument is apt to assume a prominence at the expense of the medical man. Thus it happens that an apparatus is bought by some unqualified person as a commercial speculation. It is put in charge of an attendant, dressed, perhaps, to look like a nurse, it is advertised freely in every possible way, the patient comes, takes his place at the machine,

the switch is turned on, and the attendant has only to stand by and tell fairy tales of wonderful cures to the eager patient while the machine provides the rest. The tendency is a dangerous one, and both medical man and patient are much more likely to lose than to gain by the establishment of these institutions. I was very much struck, on reading Mr. Morgan's presidential address to the Medical Society in October, 1900, to see that he expressed exactly the same opinion. He says: "The further development of X rays to surgery and medicine is a study which I should like to see appropriated by members of the profession. I believe that it is still in its infancy, and that there is a great future before it; but in order that we may obtain the full advantages of its resources, it appears to me that the study must be pursued by men having a thorough medical training, and must not be allowed by them to fall into the hands of mechanics, lest its progress as a scientific aid to diagnosis be checked. I heartily commend it as a new form of speciality to those who have time and opportunity to pursue this entrancing study." His views are already amply confirmed. The medical man is discouraged from taking up X-ray photography because he is averse to competition with the chemist round the corner, and X-ray work is languishing a good deal on that account. Not only so, but the taking of photographs by means of X rays is only the first step. The treatment of lupus by the same persons follows almost as a matter of course, and by no means should the medical profession countenance so serious a matter as the treatment of lupus by any irresponsible person who happens to have an induction coil. With the possibility of the use of Röntgen rays in the treatment of cancer the danger becomes even greater.

Much more might be said about the numerous topics of interest connected with electro-therapeutic work, but I venture to hope that in what I have already said there may be matters deserving of serious thought.

THE incompatibility of paraldehyde and potassium bromide has not attracted the attention it deserves. When prescribed together the oxidising properties of paraldehyde, according to Brissemoret, convert potassium bromide into bromate. A full account is given in the 'Journal des Practiciens' for January 18th.

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A CLINICAL LECTURE

ON

SOME REMARKABLE FORMS OF INJURY TO THE EYE.

Delivered at the Medical Graduates' College and Polyclinic, January 29th, 1902.

By HENRY POWER, F.R.C.S.

GENTLEMEN,—You will remember that a French satirist wrote a poem which he termed "Jean qui pleure et Jean qui rit," or "The man who smiles and the man who weeps." I wish to draw your attention to the fact that it is better in ophthalmic practice to be cheerful than to be sad. It is necessary in most instances to be hopeful with eye patients; they are, in a very large number of cases, very depressed in regard to the condition of their eyes; an exaggerated feeling holds them, and they think that they are likely to become blind; and so in all affections of the eye it is better generally to assume a hopeful and reassuring tone. It is difficult, perhaps, in many other affections besides those of the eye to preserve quite the proper attitude of mind with which we should deal with patients; to be grave without being austere, to be cheerful without levity, to be hopeful and confident without presumption, and to be friendly without being familiar are points, I think, that we gradually learn as we get older, and sometimes with sharp rebuffs. But the exaggeration with which many people regard affections of the eye is due largely to the circumstance, I think, that very small injuries often produce a good deal of distress. A man knows that a minute fragment of sand or coke in his eye sets up considerable distress, and therefore he thinks whatever is worse than that must be very serious indeed, and I have at times met with cases, one or two of which I will mention to you, which showed this exaggerated feeling to a very remarkable extent. A man came to me who told me he had pur-

chased or rented a certain moor in Scotland, and on rising early on the morning of his first visit, that he might go out shooting and try his luck, he walked a mile or two from the house and saw a bird, fired at it, and was, as he said, instantly struck blind. He was in a dreadful state of depression. He said he called and shouted but no one answered for about half an hour. At length a lad came up and said, "What is the matter, master?" The gentleman asked the lad, "Have I blood running down my face?" He said, "No, you have no blood." He then said to the lad, "I am blind, give me your hand and lead me to the nearest railway station." He came straight to London and I saw him late that night. He was in great distress of mind and assured me he was perfectly blind. He had a bandage on his eyes and his handkerchief was wrapped round his head. He had never opened his eyes from the time the accident occurred. I took off the bandage and examined his eyes, and there was nothing abnormal to be seen. When he looked up I said, "Can you see the clock on the shelf?" He said "Yes, perfectly." I closed first one eye and then the other, and he assured me he could see perfectly; there was nothing the matter. The man had had a fright. Perhaps he may have been struck a slight blow, but it was evidently an exaggerated feeling, perhaps inducing ptosis, which led to an apparent extinction of the sight. We must take into consideration the alarm of patients under various conditions. A gentleman æt. 80 came to me who was one of the very first explorers in that part of Africa where we are now at war. He made his way up country and saw the first Zulu army, forty or fifty thousand strong. Having accomplished his work there he came home, and required the operation for cataract extraction. I did the operation for him, and he bore it bravely without cocaine or chloroform. But about two o'clock in the morning his daughter came to me and said her father had become insane. I went down in the cab which had brought her to me, and when I reached the house I noticed not only his house but the one on either side was illuminated. I found the old gentleman lying on a bed, violently struggling with three men, one on each arm, and the third holding his legs, and there were two or three women in the room. I thought the eye was hopelessly gone.

I took the men by the shoulders and said, "Leave him alone with me for five minutes and go out of the room." He ceased to struggle. I took off his bandages and said, "Do you know who is speaking to you?" He said "Yes, quite well." I said "Do you know what is occurring?" "No," he said, "except that I have had a dreadful nightmare, and I thought I had been struggling violently." I said, "You are not struggling now; I shall put on one bandage and then you must go to sleep." Utterly prostrated by his fatigue he lay down on his side and was asleep in ten seconds. I let the people into the room again, and each one on entering said, "Is he dead?" so sudden and quick was the change from the violent struggling to the succeeding calm. He did exceedingly well afterwards, and eventually he had capital vision. That case suggested to me a good deal in regard to the bandaging of the eyes of old people who have had cataract extraction. You should be a little careful as to the nature of the treatment in them. I venture to mention another case of a somewhat similar kind, in which the result was not so fortunate. It was the case of an old lady, and I did improperly under the circumstances. She was eighty-two or eighty-three years of age, and if I were to mention her name, it would be familiar to every one in this room. She was quite deaf, and also very nearly blind from double cataract. I operated upon one eye, and the operation went off exceedingly well; there was no difficulty, but I injudiciously put a bandage upon both her eyes. That procedure resulted in the loss of the eye operated on. She was, as I said, perfectly deaf, and time hung heavily upon her hands. She was very good for the first day, and was fairly restful during the second day, but the third day was too much for her. She wanted to ascertain whether she could see or not, and taking up a knitting needle, she plunged it under the bandage and tilted it up, and in so doing, burst open the wound and the eye was lost. I was exceedingly sorry for her, because I understood that my having put bandages on both her eyes made the time unbearably long and dull for her.

I will tell you another case of exaggerated sentiment about the eye. A clergyman came to me from Wales stating that he had been suddenly smitten with a ray of light which had blinded him. He presented himself with goggles and bandages

over his eyes as well as a great shade over his brow, and everything was done to shut out the light as much as possible. He said he lived in the Salisbury Hotel, Fleet Street, and asked me to go and see him there. I found him in the deepest darkness; he would allow no ray of light to come in. I could see nothing wrong with his eyes, and so I asked him what the ray of light was. It proved to be a reflection of the sun which he had seen upon an ivy-clad wall. It was therefore not a very strong reflection, but it seemed enough, in the particular condition of mind that he was in, to impress him with the idea that his vision was lost. That old fellow got quite well. I went down week by week to him, and I could tell what his conditions were by the state of the blinds in the room which he occupied. When the blinds were fairly high up he was better; when they were down completely he was worse. That went on for a considerable time, but at last, under the use of strychnia, he got well.

Such cases are not very uncommon, and are the result of exaggerated impressions of trouble in the eye, or proceed from the idea that something serious has affected the sight. There was another case which I might mention, the patient being a cousin of a well-known physician in the south of England, who brought him up to me some years ago, and lodged him in an hotel near Westminster Abbey. The gentleman, æt. 50, was said to have gone blind. When I went to see him he was in bed, the room was exceedingly dark, and I could not see where the bed was. I put out my hand and found his hand was warm and comfortable, and put my hand to his face and found his eyes were not tender to the touch. I tested the tension roughly and found it was normal, and as far as I could tell there was nothing particularly wrong about him. I asked that a lighted candle should be brought in, and when this was done he said he could see it. I thought it was a case of mental disturbance, and I therefore patted him on the back and said, "There is nothing the matter with you, get up and go to the theatre." He rose, and recovered; there was no further trouble with him. There are some cases in which firmness is required.

I now wish to speak of some unexpected forms of recovery after severe accidents to the eyes. I think the first one that impressed me very strongly

was that of a cabman who was brought into St. Bartholomew's Hospital with a very severe injury to the eye. Upon examination the conditions present were very serious. He had been knocked off his seat by a collision with another cab, and the result was the bursting open of one eye on the outer and lower part. That wound was not only unusual in position, but it was also an extremely severe one. The eye was ruptured with a lacerated wound, the vitreous was protruding, and the choroid and retina were visible. If it had been that the man was an ordinary case I should have said directly that the eye must be removed. But it happened that upon examining him we found he had impaired vision with the other eye. Considering his occupation we thought it would be expedient to try to preserve the vision and to postpone enucleation. He was a very good-hearted fellow. The wound was of a jagged description, and apparently the eye was utterly lost. However, some very fine sutures were used and the edges brought together, which, in consequence of the collapsed condition of the globe, was not very difficult. The jagged irregular conjunctival tissue was brought over it, and so it was converted into a sub-conjunctival wound. Union took place quite easily, and the recovery of the patient was so considerable that he was able to get about the street quite easily. He afterwards told me that he could drive his cab, but he was afraid to do it, because, only having one really good eye, he was afraid that some accident might occur. But his recovery of vision was sufficient to enable him to get about safely.

Another class of cases are those in which severe blows have been received. The best case I can remember at the moment is that of a well-known musician, who was organist of St. Paul's Cathedral. He was playing at tennis at Cirencester, and his friend hesitated over a ball; he turned round to see what he was doing, and at the same time received the tennis ball full upon the left eye. Unfortunately for him the sight of his right eye had been so far lost that he had only perception of light with it. In the left eye he had previously had very good vision, but the sight was instantly utterly abolished, so that he was unable to see even the light of the sky. He was dreadfully distressed, and came straight up to London, and I saw him an hour or so after his arrival here. I

thought the eye was entirely lost. There was no decrease of tension, and therefore no rupture of the globe, and there was no blood in the anterior chamber, but on looking behind the lens with oblique illumination, and especially with the ophthalmoscope, one could see great drops of blood one behind the other for some distance. Either the shock or the discharge of blood was so abundant that he was quite unable to distinguish the light of a candle. The question was what was to be done for him? He was exceedingly anxious to know whether it was possible for him to recover any vision. I think in that case a cheering forecast was of help. I told him it was possible that the blood might undergo absorption, and that he might once more recover his vision to a limited extent. He consented to be put to bed, black hangings were placed before his door, and he remained perfectly quiescent for nearly three weeks. I told him to lie either on his left side or his right side, but that he had better not lie straight on his back with his head back, because I thought it possible that the blood might gravitate to the bottom, and when the hæmoglobin had been absorbed there might remain a good deal of fibrinous material, which might form a thin layer. It was to prevent this that I told him to lie either on one side or the other. At the end of the three weeks I went into the room with a lighted candle, and to his astonishment and delight, and mine also, he said, "I can see the candle." Upon examining the media I found them pretty clear. He was very chary about having the eye examined, because it was excessively tender and irritable to light; therefore I did not make a very detailed examination, and I never did ascertain where the blood came from. But certainly the vitreous was completely infiltrated with it at the time of the accident, and it had vanished in three weeks. That is a very interesting case, and I can only add that although he found the work at St. Paul's too severe for him, and therefore gave it up, he went to Magdalen College, Oxford, and for some years did his work there as organist, and had no difficulty about his ordinary duties.

Another case was one in which in a serious accident I think hope did a deal of good to the individual. I was sent for to Woolwich to see a student who was just going into the college, and was said to have blown himself up with gunpowder.

I found a young fellow lying on a sofa with his mother beside him bathing his head with water. The young man complained of very little pain. It transpired that he had not been playing with gunpowder, but with fulminate of silver. He had made about one and a half grammes of it in a small porcelain capsule, and when dry had taken his pencil and very foolishly stirred it up. It immediately exploded, and the violence of the explosion saved his eyes, paradoxical as that may at first sight appear, for what had occurred was that the substance was for the most part reduced to extremely fine powder. It affected both eyes, and when I opened them each cornea was covered with an exceedingly thin layer of white porcelainous dust. I was able to pick off from the surface a good many fairly large bits without using cocaine and without much trouble. But when I had got out all I could remove the surface still remained perfectly white. Remembering the difficulty which is always felt in maintaining the blackness of tattoo in various cases of leukoma, I said it would probably clear away in time, but that it would be many months before this would happen. That forecast was incorrect, because it cleared away very much sooner, and in less than three months he came up to me with a tolerably clear cornea, and he was able to resume his work. That occurred about ten years ago, and only the other day the patient called on me and asked whether I remembered him. He said, "I was the young fellow whom you saw because he had blown up his eyes with fulminate of silver." Then, of course, I remembered the case on account of its unusual features. His eyes were now perfectly clear and natural. I think hope did a great deal for that young fellow by leading him to think that recovery would eventually take place.

There are certain cases of burns which are very instructive. What should be done in cases of burns? Not very long ago I was asked to see a lady residing near me. The mother said, "I fear it is a bad case, for my daughter was curling her hair with hot irons when they slipped, and her eye is burnt." There was a great deal of pain and swelling, and I must say I took a very unfavourable view of it. I thought a hot iron applied to the surface of the cornea would produce a good deal of alteration in it. I examined her eye, and although it was very tender I found a white band

as broad as a pencil across the front of the cornea. I thought the eye would be permanently marked. I instilled some cocaine, and put the patient to bed with a cold lotion upon the surface, and ordered her a morphia pill to enable her to have a good night's rest. The next morning, to my great astonishment, the eye was as clear as mine. The hot iron must have just seared the surface, and in some way the superficial cells had probably been washed or cleared off by the tears. But no one could be more astonished than myself at finding that the distinct band of burnt tissue had disappeared. What makes the case more curious is that in the case of various escharotics a very different result must be expected. I have in my time seen burns from hydrochloric and nitric and sulphuric acids, and in many cases the effects have been disastrous; but none of these have been so bad or so serious as those cases in which mortar or lime has entered the eye. In my own experience the entrance of lime into the eye is fatal to it. If an eye so damaged were at once washed out with a little milk or oil or some weakly alkaline or bland fluid the eye might recover, but under the ordinary conditions of life, where perhaps half an hour elapses before anything is or can be done, and where the lime is absolutely embedded in the cornea, you must take a hopeless view of the case. I would not recommend hope to be put forward in cases of that kind. Some here may have seen bad cases where molten metal has gone into the eye, and those are very extraordinary cases. You will remember the particular proceeding known as making a Boutigny's button. If you take a red-hot shovel and pour a drop of water upon it, the drop will roll about and will not disappear until the shovel has got cool enough for the shovel to come into contact with the water. The reason is that on the red-hot shovel the water gets surrounded by an atmosphere of vapour, which is a bad conductor of heat, and the drop only slowly evaporates. It moves about like a globule of mercury, and that appears to be what occurs in many of these cases. I have seen three or four of them where men have had splashes of metal falling into their eyes. Very often plumbers suffer from such splashes, and the lead is found to have moulded itself upon the surface of the eye, and the lids have been covered by it, and when the eye is opened a thin fragment can be seized

and pulled out, the cornea being quite transparent beneath, and no damage has been done at all. I imagine that the vapour of the tears is on either side of the metal, and the burning effect is prevented.

All burns from alkalies are very serious, much more serious than the acids. I have seen many from alkalies, particularly caustic alkalies, and all the cases have led to permanent impairment of vision either from leukoma or symblepharon. They seem in most instances to almost tan the connective tissues and convert them into cicatricial material, which can be pulled out in shreds with a pair of forceps, and is apt to contract. But it is hopeless to think of obtaining a clear cornea in such accidents.

Then there are certain cases in which fibrinous iritis occurs. I remember one in St. Bartholomew's Hospital which I was hopeless about, but I think I ought to have been hopeful. The patient was a waiter, and when he came in he had a very small pupil, but the surface of the eye was blue, and seemed pretty natural. In the course of a day or two, whilst he was in the hospital, he had an attack of acute fibrinous iritis, and I think that is about the only genuine case of the condition I have seen. The whole surface of the eye was covered with greyish material like chamois leather, but I did not recognise the nature of the disease. The patient had given to him some mercury, and afterwards iodide of potassium, and gradually the deposit cleared off, and left the eye just as it was before, with very good tension. That was a case where hopefulness would have been of much service in the first instance and where I should have practised it.

There is yet another class of cases in which good results sometimes belie our anticipations—I refer to those of interstitial keratitis. The symptoms of that disease are familiar to all from Mr. Hutchinson's masterly descriptions of them. Let me record one such instance. A girl æt. 10 was brought to me with hazy corneæ, and the usual marks of inherited syphilis. Mercury in several forms, potassium iodide, quinine, and strychnia and iron were given internally, and atropine blisters and various other local remedies applied. No improvement took place, but rather deterioration. Both corneæ became perfectly opaque, and she only had perception of light. I afterwards heard that at

this time she was shamefully neglected by her parents, who both drank heavily. Her food was sparing, and often uncertain, and she was frequently beaten. She continued practically blind so long that I recommended she should be taught to read on the Braille system; fortunately for her her parents died, and a relative charitably devoted herself to her welfare, both bodily and mental. She responded to that kind care, improvement commenced, and now, after the lapse of ten or twelve years, she can read No. 8 of Jäger's test types, and possesses very useful vision for all purposes.

Then there is the long series of gun-shot wounds, and that is where the treatment requires to be very carefully determined, and much thought is required in each case. When a large shot strikes the eye, and ruptures it clean across, I think you can do nothing; the lens is generally seriously damaged, and the vitreous and iris and adjoining parts are injured, and the eye is practically useless. But there are many cases in which a small shot penetrates the eye, and may either be lodged in the sclerotic or may pass right through the eye. This, I think, should not involve the removal of the globe. There was a man whom I was sent for to see near Tunbridge Wells, and I think I acted injudiciously with him. He was in process of building a new house, and one day, when walking along a ridge, he saw a rabbit. His son was walking just below the ridge at the same time and also saw the rabbit. The son fired just at the moment that his father's head appeared above the level of the ridge. One or two pellets penetrated his eye, and apparently it had seriously damaged his vision. Influenced by having previously seen one or two cases with Sir William Bowman, who invariably said you had better remove the eye and then there is no danger of sympathetic ophthalmia, I removed the eye. But I think that in this case the shot traversed the eye and was lodged in the connective tissue and fat in the orbit, and in view of the subsequent examination, I do not think it would have done any harm if I had allowed it to remain, for on careful dissection I was exceedingly sorry to find that there was no shot in the eye. I have seen other cases where a shot has gone into the eye, and I think you should wait until symptoms arise before you proceed to so serious a course as enucleation of the globe.

There was a man, a surgeon, who lived in Holborn, and he was injured in very much the same way. But being a surgeon he was very prudent, and said, "I will not have anything done, you may pick out what shot you can find, but I will not have the eye removed." In his case I removed shot from three positions, one of them being embedded in the sclerotic, the others in the lids. There was no pain or trouble about the removal, but there was the mark of one shot which had, I think, penetrated the globe; and in that case, I have no doubt, if the patient had not been a surgeon, as there were several other shots present, I should have removed the eye. That case did well, he had no trouble from it, and for several years he had proper vision, and I had no necessity to perform any operation. So I would say that in gun-shot wounds it is desirable to be cautious about operating. Mr. Berry will probably recollect a very well-known case, which I reported some time ago, of a young fellow who was shot by a friend, though not in a very friendly proceeding, in the inner side of the eye. The bullet penetrated on the left side near the caruncle. The immediate results were not very clear; he did not lose his sight entirely; he retained some vision, and had a good deal of hæmorrhage at the time. That man, after a lapse of some weeks, began to develop an aneurysm at the inner side of the eye. In that case it was exceedingly difficult to determine what should be done. It was so difficult that the ophthalmic surgeons thought it worth while to ask the opinions of the staff generally, and among the opinions we obtained on the matter were those of Sir James Paget and Sir W. Savory. We were very much interested at the difference of opinion which was expressed by different surgeons. One recommended that a cut should be made and that the aneurysm should be explored. When we came to think it out it appeared a formidable proceeding. The aneurysm, in all probability, was due to some lesion of the ophthalmic artery. If not, it was possibly some enlargement of the carotid artery. If we had cut down upon it and had some sharp bleeding, we might not have been able to stop it, and death might have ensued as a result. Others thought that pressure could be adopted, and that was practised for some time, and pressure was also exerted with the fingers upon the carotid artery with the view of stopping the

development of the trouble. But that proved so irritating that the patient declined to have it continued any longer. So after many days of thinking what we should do, tying the carotid was forced upon us, for one night hæmorrhage took place with great violence from his nose, and he lost a quart of blood. He was thin and delicate originally, and this loss of blood reduced him to a condition of perfect anæmia, so we thought it was wise to tie the carotid artery. The parts were exposed, but it was very difficult to tell whether we had the pneumogastric nerve or the carotid artery before us. Both were white, and no pulsation could be perceived. Perhaps our anatomy was a little at fault, but we were not quite sure. My son looked at it, and we determined that one was the artery, and so it proved. The result of the ligation of the carotid was that the aneurysm stopped increasing, and there was no further trouble or hæmorrhage; in the course of two or three months he recovered. I saw a lady who knew him some time afterwards, and she said he was nearly blind with that eye, but he was doing his work as a banker in New Zealand. That case opens up a serious question as to what should be done in the case of gun-shot wounds in the inner part of the orbit.

There was another case of a similar kind where a woman was lying on a sofa. Her son had recently returned from America and was playing with a toy pistol, when it suddenly exploded. His mother gave a scream, and it was found that the shot had struck her on the inner side of the eye. In that case, although it produced blindness of that eye, it did no further damage to it, and I think the bullet dropped into her stomach, because she felt something fall immediately after the accident and the bullet was never found. At all events that was the termination of that case. She lost her vision in one eye, but nothing was or could be done for her except to keep her in bed and at rest, and under as favourable conditions otherwise as possible.

Then there are cases, of which I have seen several, where glass gets into the eye. It is very difficult to know what to do with such accidents. In some, of course, the eye is lost. A young fellow came to me the other day whom I saw twenty years ago. In making a hydrogen flask explode, one eye was lost, and he has never had vision with

it since. I had to remove the atrophied globe not long ago. Another case which I remember was very interesting in regard to treatment. It was similar in character. A man had a blow from a broken flask, and a fragment of glass presented itself in the pupil when the eye was quiet. We could not tell how far it extended behind the iris. It was evidently a large angular fragment, but it had not damaged the lens or touched the iris. It had introduced itself between the capsule of the lens and the uveal surface. What should be done in a case of this kind? It was quite quiet, the man had no pain (he was a medical man's son), and we left it alone. He used to come periodically to the hospital (once a week for several months) to show himself. He remained with a fair amount of vision for about one and a half years. At the end of that time he had to go to Australia or New Zealand, and having come to that determination the question arose whether it was wise for him to go with glass in his eye and run the risk of being knocked about on board ship by bad weather, or possibly get it irritated and inflamed when he had arrived at his destination, where he could not, perhaps, get proper attention, or whether it would be better to make an attempt to remove it. I told him that if any attempt were made at removal it would probably lead to loss of the eye. He decided to have it done, and I am afraid the result was as anticipated. We were able to seize the fragment of glass, but the result was a slow inflammatory trouble, which led to loss of vision. Perhaps he would have done better to leave it in the eye, except, perhaps, for the fact that he might have had sympathetic ophthalmia supervening in the sound eye.

Besides the intense nervousness of patients, of which I have said something, and unexpected recoveries, we now and then meet with cases of what may be termed mistakes in diagnosis. I suppose we are all of us familiar with mistakes, and, of course, mistakes in diagnosis make a great deal of difference as to what is done for a patient. A lady came to me who had a slight swelling at the sclero-corneal junction of one eye. The swelling complained of was easily removed and no further trouble was anticipated, but about three weeks afterwards she came to me again with a recurrence. It had extended, but was still small. I then said, "We must remove this for you a little more freely."

I did so, carefully scraping the surfaces and applying iodoform, and afterwards perchloride of mercury, hoping it would by that means be cured. But by-and-by there appeared in the anterior chamber a recurrent round-celled sarcomatous growth. I am afraid I operated upon her five times, with the result that the globe and contents of the orbit had to be removed. Ultimately that patient died from cancer in her liver and other organs; indeed the cancer seemed to be distributed throughout her whole body. Very shortly after another lady came to me who also had just such another pale pinkish growth in the identical region. I was rather shy about touching that, bearing in mind the previous case, and I thought I had better leave it alone. But she found it was growing and she said, "I will have it removed whatever may be the result." I removed it for her, and she had no further trouble; the parts healed quite well and there was no recurrence. It was clear that I had made a mistake in the diagnosis, because I thought it was a sarcoma and that it was very likely to recur. Those cases just show that sometimes when you think you have a bad one it turns out all right, and, on the other hand, what seems to be a favourable case may not prove to be so. There are certain cases in which we ourselves make the wound, and a long delay takes place in recovery. I would recall one or two cases; such, for example, as those where after operation for cataract the wound fails to unite. I remember one under my care at the Westminster Ophthalmic Hospital which was very remarkable. The patient was a feeble old lady, and the operation was done with the triangular Beer's knife. Apparently everything went on well. But in her case a little blood was effused and the edges of the wound failed to unite. I had her under my care probably a month, and at the end of that time the edges of the wound were still apart. Charles Guthrie was alive then, and he said, "I will make the edges unite." He took a stick of solid nitrate of silver and touched the whole surface of the cut edge freely with it. It did unite, and the eye was very good afterwards; but I thought that was a rather dangerous procedure, and I confess I should not like to repeat it.

In regard to mistakes in diagnosis, there are many which you will find referred to in the books. Only a day or two ago I was looking through

Yvert's treatise; he mentions a case in which every symptom of cancerous growth existed at the back of the eye. The eye protruded and was painful, and there were nodulated growths felt all round it. He concluded that the whole globe would have to be removed, and the orbit eviscerated. The old gentleman, who was a homœopath, declined to have anything done with the knife. Whilst the matter was being discussed a slight rupture took place, and a large quantity of pus was discharged. After that the man recovered, and there was no further trouble. It was merely an abscess. That is very suggestive. You may sometimes deal with cases of that kind hopefully, though at first sight they seem as if they will certainly be disastrous.

Then there are cases, of which I have seen examples, where the disease was of a syphilitic type. I very well remember a case at St. Bartholomew's Hospital where there was a tumour which we all thought was serious in nature, but under the influence of iodide of potassium and a little mercury the man entirely recovered.

The point I have been endeavouring to impress upon you this afternoon is that there are many cases which present themselves which are to be treated hopefully, that you are not to go to a patient and at once give him cause for depression because his condition appears to be serious. Nature, as Goethe says, is a kind mother. The tissues of the eye have remarkable recuperative power, and cases that at one time seem hopeless, occasionally, like some of those I have adduced this afternoon, recover excellent, or at least very useful, vision.

Mental Development of Cretinous Child under Thyroid Treatment.

—Heller states ('Wiener klin. Rundschau,' February 2nd) that he has never seen in the literature the announcement that thyroid treatment has been systematically employed in schools for mentally deficient children to transform untrainable into trainable subjects. He thinks that the combination of thyroid with pedagogic treatment promises better results than hitherto attained, and describes a case in detail to illustrate the unexpected advantages derived from this combination in treatment of a supposed hopeless cretin and idiot.—*Journ. A. M. A.*, March 8th.

A SURGICAL DEMONSTRATION

At the Medical Graduates' College and Polyclinic,
January 29th, 1902.

By JAMES BERRY, B.S., F.R.C.S.

GENTLEMEN,—There are several cases here to-day, none of which are very rare or unusual, but they illustrate various diseases that we meet with frequently in practice, and therefore I hope they may prove to be of interest to you. The first case is that of a young man who has been sent here by Dr. Gabe on account of a swelling in the neck. I begin by asking him how long he has had this trouble, and he replies two months. The first thing he noticed was a swelling on the right side of the neck. It has gradually been getting larger. His health has previously always been good, and he has had no affection of the ears, throat, nose, or face. The first thing I notice about him is that he is pale and rather thin, and that he does not look very strong or robust. Otherwise he seems to be in very fair health. I find on the right side of his neck a considerable swelling, which is obvious to all of you. Bending his head down so as to relax the sterno-mastoid muscle, I find the swelling is fairly moveable upon the deeper parts. On putting the sterno-mastoid into action, the swelling is found to be mainly underneath that muscle. It is irregular in shape, and evidently consists of a mass of smaller swellings more or less loosely united together. There is some redness of the skin over it, and at one spot it is soft. There can be no doubt that it is a mass of enlarged cervical glands, and that one of them is suppurating. On the left side of the neck there are also several more enlarged glands. There are also some at the root of the neck on both sides and in both axillæ.

We have now to seek the cause of this glandular enlargement, and we must naturally examine all those parts from which these glands receive their lymphatics. On looking into the mouth I see at once what is certainly enough to cause this trouble, namely, many carious teeth. There appears to be no other source of infection. The diagnosis is obviously enlargement of lymphatic glands, due to absorption from carious teeth, and probably tuberculous in nature. So far as I can tell, from a somewhat cursory examination, there is no evidence of tubercle in the lungs or elsewhere. This is a point to which attention should be directed.

Now about treatment. The first thing to be done is to attend to the teeth, and by stopping or extracting them, to prevent further infection. The general treatment may comprise the administration of such drugs as cod-liver oil, steel wine, or iodide of iron. With regard to the glands themselves, I think there cannot be much doubt that it is too late now to prevent suppuration. This region, where the skin is thin, red, and evidently about to ulcerate, should be incised, scraped, and dressed with scrupulous attention to cleanliness. With regard to the glands that have not yet begun to suppurate it would be well to watch them for a week or two before saying whether it would be advisable to remove them or not. They are very widely distributed, and it would be impossible to get the whole of the disease away by any operation. Therefore I should be inclined to deal at once with this gland which is on the point of discharging externally, and to leave the others for the present. After the treatment which I have sketched to you it is possible that the enlargement of the other glands may subside. One often sees a considerable subsidence of tuberculous glands after treatment of the primary source of infection. My reason for not recommending an operation for the removal of all the affected glands is that the disease is so widespread that complete removal would be impossible. Possibly one or other of these glands may subsequently suppurate and may then require to be dealt with by erosion or excision.

The next case is that of a man æt. 41, who comes on account of a sore place on the side of the nose near the root. It is said to have been in existence eleven years, and to have begun as a little pimple. The patient gives a history of its having gone away for several years, and then returned. He says that three weeks ago it recurred, and he has since been scratching it with his finger. The man, however, is a foreigner, and is unable to speak English. The history, therefore, which is obtained through a friend, is, I am afraid, not very reliable. You see a sore which is comparatively dry, and surrounded by a red ring, the edge of it being very slightly raised. The diagnosis lies between rodent ulcer and some quite simple inflammatory disease, possibly an ulcer, the result of an accident, which has been subsequently scratched. The situation of it is very suggestive of rodent ulcer. These ulcers are most often

found in the upper half of the face, and are especially common about the eyelid and side of the nose. The main point of distinction between a rodent ulcer and a simple form of ulcer is the presence of a definite growth. Rodent ulcer is a slowly growing form of carcinoma, and there is always a certain amount of new growth, new tissue at its base. But rodent ulcer differs from all other forms of carcinoma in that the ulceration takes place almost as fast as the growth, so that at no time is there much actual new tissue present, though there is always some. You will always find around a rodent ulcer a slightly raised margin. In this case there is not as much raising of the edge as usual. I certainly feel extremely suspicious that we have to deal here with a rodent ulcer. There is an element of doubt in the case on account of the somewhat contradictory history, and the fact that the ulcer itself is not quite typical. The ordinary history of rodent ulcer is that it begins as a little pimple, and very often in quite early adult life, and after many years' existence it ulcerates, a scab forms upon it, and it refuses to heal.

The next case is that of an old man *æt.* 70, whom I saw for the first time in September last. He was then transferred to my care by one of my colleagues at the Royal Free Hospital. The history was that nearly two years ago he first had an ulcerated lump on the side of his cheek, just in front of the ear. This grew steadily, and became somewhat prominent. About a year ago he underwent an operation at some other hospital for removal of this ulcer. This was followed by paralysis of the upper part of the facial nerve. The ulcer recurring, my colleague removed it freely, taking away the whole of the skin over an area half the size of the palm of one's hand. In the course of the operation the whole of the facial nerve was divided and partly removed, quite rightly, because it was involved in the growth. You will notice that on the left side there is marked ectropion. We are told that this has existed since the first operation a year ago. It is evidently due to paralysis of the upper part of the facial nerve, and is not due to contraction of scar tissue, because there is no evidence of any scarring about the front of the cheek. After the second operation, which was a very extensive one, the healing of the wound was effected by the employ-

ment of large grafts of skin. You will notice now that there is a small indurated and ulcerated area at the anterior and upper part of the scar. This is evidently a recurrence of the original growth. As to the diagnosis, I think most of you will be inclined to agree with me in saying that at first sight this looks like a rodent ulcer. The man's age, the situation of the ulcer, and the presence of the raised margin, all favour that diagnosis, but the short history and the prominence of the growth are unlike rodent ulcer. It is very unusual for rodent ulcer to attain a large size within the space of two years. In this case there is no doubt about the diagnosis, because the growth was removed and examined microscopically, and it was found to be epithelioma. Of course there is all the difference in the world between rodent ulcer and epithelioma, although I have occasionally heard surgeons speak as if they were one and the same disease. The characteristic feature of rodent ulcer under the microscope is the epithelial growth extending outwards beneath the healthy skin, in a manner not seen in epithelioma. In the latter you find the epithelium growing down from the surface, and not extending subcutaneously as the rodent ulcer does. It is the subcutaneous element in the rodent ulcer which makes the disease so apt to return after ordinary removal. If in removing rodent ulcer you carry your knife round just outside the edge of the visible growth, you will certainly carry it through the subcutaneous part of the growth, and will consequently leave some of the tumour behind. That is why it is so important in removing rodent ulcer to take away at least a third of an inch, or better half an inch, of surrounding apparently healthy tissues. When I was, some years ago, a surgical registrar at another hospital, it was my duty to examine microscopically all the tumours that had been removed by operation, and among them many rodent ulcers. It was not uncommon to find microscopic evidence of the growth characteristic of rodent ulcer at the periphery of the portion of skin that had been removed, and this even when, to the naked eye, it seemed that the skin there was perfectly healthy. In other words, removals that seemed to be complete and satisfactory were proved by the microscope to have been incomplete. The occurrence of rodent ulcer upon an exposed part like the face naturally makes the surgeon wish to remove it in

such a manner as to cause as little disfigurement as possible. For instance, when it occurs on the inner canthus of the eye there is a great temptation to save as much as possible of the eyelid. But I cannot help feeling that it is wrong to remove rodent ulcer by cutting very near to the visible disease, because if that is done the operator is sure to leave behind invisible portions of growth, and this leads to recurrence. It is better at the beginning to perform, if possible, a free removal of the growth.

In the case before us the disease is true epithelioma, which is much more malignant than rodent ulcer. It is characterised by a much greater amount of new growth than is found in a rodent ulcer.

The next patient I have to show you comes complaining of swelling at the back of his right knee. You will notice that over the inner part of the popliteal space there is a swelling as large as an egg, and over it a long scar, and some of you may be able to perceive the suture marks on either side of this long, broad scar. Obviously it is an operation scar. First of all I draw your attention to the fact that, although that scar is half an inch wide, nevertheless it is the result of a wound which has apparently healed by first intention; I say it healed by first intention partly because the suture marks are very distinct, showing that the stitches were left in for several days, also there are no signs of any part of this scar being puckered or depressed or thicker than the rest. It is thin and supple throughout. Wounds that have healed by first intention, and that present at first a narrow linear scar, will often, after months or years, show a scar that has widened from the stretching of the scar tissue. Therefore, the mere width of the scar in this case is no proof that the wound did not unite by first intention. The man tells us that he has had the swelling eight years, and that it gradually got worse until a year ago, when he went to a London hospital and had an operation for its removal. He left the hospital two or three weeks later, apparently cured, but after three months he found that the swelling was coming back again, and accordingly he returned to the hospital. It was then punctured, but he does not know what came out of it. After that, the swelling disappeared for a few weeks, and then it began to return, and it has got bigger and bigger. He now comes to

another hospital, asking for further treatment. You will notice that there is an oval soft swelling situated at the inner side of the popliteal space. When he bends the leg you can see the semitendinosus, and you can feel the semimembranosus. The swelling is situated just outside the semimembranosus muscle; it is just between the semimembranosus and the head of the gastrocnemius. When he flexes the knee fully the swelling disappears. It is, in fact, a semimembranosus bursa. These bursæ often communicate with the knee-joint; therefore, it is well to examine the knee and see if there is any sign of disease there. It may be that the man has chronic synovitis, and that the fluid runs from the knee into the bursa. I have examined the knee carefully, but cannot find any fluid or other evidence of chronic synovitis. With regard to the treatment of this bursa, as it gives him a good deal of trouble, and he is very anxious that I should make an attempt to remove it, I propose to cut down upon it and remove, if possible, the whole sac. These semimembranosus bursæ are sometimes difficult to remove completely, on account of their connection with the posterior ligament of the knee, and the manner in which they are blended with the tendons. If the whole sac is not removed, the disease is very apt to return. [A few days later the bursa was dissected out. It was found to communicate freely with the knee-joint, and to have two deep prolongations extending upwards and downwards among the muscles and tendons. These had apparently escaped notice at the first operation.]

Our next patient is a girl æt. 17. She comes to hospital on account of a swelling of the knee, which has been present for several weeks, but there is very much less swelling now than when I first saw her a week or ten days ago. I may say at once that that swelling was in the knee itself, which was full of fluid. It was a case of synovial effusion into the knee-joint, and even now you can see there is a general swelling in the situation of the synovial membrane. There is no swelling over the patella, but around the patella there is the usual horseshoe swelling of the joint. When I put my hand above the knee and push the fluid downwards I find that the patella rides, showing that there is still some fluid in the joint. The point we have to discuss is the cause of this effusion of fluid. The history does not help us

much ; she has never hurt the knee, and there is no history of a sudden twist or anything suggestive of a loose cartilage. When a young person has a chronic swelling about a joint one naturally thinks of tubercle, and in this case there is a rather strong family history of that disease. Her father died of congestion of the lungs and bronchitis, and my house surgeon tells me that several members of the family have had tubercle. Still it does not strike me that this is quite like a tuberculous knee ; there is more fluid than is usually found in a tuberculous knee, and the synovial membrane is not appreciably thickened. I find no evidence of tubercle in other parts of the body. Therefore I naturally look for some cause other than tubercle, but I notice that her eyes look strange, and that she is rather deaf. In the corneæ there are opacities, especially in the right eye. In the left eye there is a very slight nebula. These signs taken collectively are rather suggestive of congenital syphilitic disease. But they are not conclusive, because when you look more closely into this cornea you will find there are very distinct nebulæ, separate areas of opacity ; these are more like the nebulæ produced by corneal ulcers than the general haziness usually seen in interstitial keratitis. So this appearance should not be taken as proof that the patient has suffered from congenital syphilis. Next I examine the choroid, but I do not find any evidence of the usual mottling,—the mottled choroiditis which is so often seen in congenital syphilis. I have asked my ophthalmic colleague to examine her eyes, hoping he would tell me definitely whether the disease was syphilitic or tuberculous. He says it is a very interesting case, but he will not express a definite opinion. So we cannot accept the evidence afforded by the eye as throwing any strong light on the nature of the joint condition. The conformation of the teeth does not throw any light on the matter. There is one which is ill formed, but there are none of the regular screwdriver type, and none with a central notch. I have had her throat carefully examined for scars, but found none, and the face has been looked at carefully, but there are no scars there. My house surgeon has gone carefully into the early history of the case, and finds there is a clear history of syphilis in one of the parents, and that the child suffered from a rash and other symptoms of congenital syphilis

when an infant. There is a strong probability, therefore, that the disease of the knee is due to congenital syphilis, and I may mention that since she has been under my care, during the last fortnight, she has been taking good large doses of iodide of potassium, and a Martin's bandage has been applied locally. The result of that combined treatment is that the effusion has almost entirely gone. But that does not prove very much, because it may have been that the iodide of potassium had no effect, and that the bandage caused the disappearance of the effusion, or, on the other hand, it may be that the iodide of potassium had some effect upon the syphilitic trouble, and that that was the real cause of the improvement. Therefore I am afraid we must leave the diagnosis somewhat uncertain. There is a strong suspicion that it is a syphilitic case, but there is no actual proof that it is not tuberculous. A chronic effusion of fluid into a joint is often due to some form of septic absorption from some mucous surface, such as we see, for instance, in the so-called gonorrhœal rheumatism, but the question of septic arthritis has been gone into, and there is nothing to support it.

This next patient, a woman of about forty, comes because there is something wrong with her tongue. I ask you first of all to notice the condition of her lips, which are red, and show chronic superficial inflammation. When she puts out her tongue you will see that the surface of it is exceedingly irregular, and there are a number of fissures in it and lumps about it. You will also notice that it has a red, glazed appearance. There is little or no normal epithelium on the tongue. The edge of it is irregular. I ask her how long she has had it ; she replies, ever since she was five years old. Whether it was like this at five years of age is, I think, open to doubt, but it is evident that for many years her tongue has been in this condition, because the evidences of old inflammation are very obvious. It does not take very long to decide what this ulceration is due to ; it is certainly tertiary syphilitic disease of the tongue. But she seeks advice not so much for the general condition of her tongue as for a recent trouble. On the right side of the tip of the tongue there is an ulcer which has not healed. It is obviously recent, and the patient says it has been there for two months. If you look closely at it you see a flat ulcer, and you can

feel that there is distinct induration all around it. You will perhaps think at once that I shall say it is carcinoma superadded to chronic tertiary syphilitic trouble. It may be, but I am by no means sure of it. I want to point out that the tongue is undoubtedly syphilitic, and that there is certainly a recent ulcer, but that ulcer is exceedingly flat, and its induration is not quite so well defined as that of carcinoma. I am willing to admit that it is a very difficult case, and I think I ought to say that because the surgeon who was kind enough to send the case up to me is of opinion that it is carcinoma.

My own view of the case is that the ulcer is not carcinomatous but merely a recent fresh manifestation of syphilis, and that the surrounding induration is inflammatory. My reason is that it is not quite hard enough, it is not such a definite well-defined lump as you expect to have in carcinoma, and the ulcer itself is flat, and the edge and surface are not quite those of ordinary carcinoma. It is not common to find carcinomatous ulcers in women, although one does see them every now and again in this sex. The condition of the glands must be considered. Will they help us? Here is a somewhat enlarged and rather hard gland beneath the jaw, and on the other side I find a similar one. There are also one or two lower down in the neck. I do not think they help us at all towards the diagnosis. If I had found one gland on the same side as the ulcer, I should have thought it suspicious; but when I find the glands on both sides equally enlarged, and that they are not particularly hard, I am inclined to say that they are glands that have been enlarged for a long time, owing to the chronic inflammation of the tongue. I have seen several cases of ulcer of the tongue with surrounding inflammation which had been diagnosed by competent observers as carcinoma, but which have got well completely under antisyphilitic treatment. In this case it is proposed to remove a small piece of the ulcer for microscopic examination, a course with which I entirely concur.

Here are two cases which have come from the Alexandra Hospital for hip disease. They both illustrate complications of hip disease. This boy, æt. 6, has been in the hospital about a year. The first thing you notice about him is that his left hip is somewhat flexed. There is some wasting about the muscles, and when I try to flex the leg, I find

the hip-joint is very stiff. I find that there is very considerable local thickening about the trochanter, a common and important sign of local disease. When examining a case of supposed hip disease, it is well always to examine the hip with the thumb and finger in the way that I show you, in order to ascertain how much antero-posterior thickening there is. That simple procedure gives a good deal of information about the extent of the disease. If I turn the patient over you can see the scar of an abscess, which he had six months ago. That abscess was opened antiseptically in the ordinary way, and you see that the scar is now quite sound and that there is no trace of suppuration. So far, then, this is a very straightforward case of hip-joint disease. The limb is now in a fairly good position, and the patient is practically quite well as regards the hip. But the disease of the hip is not the whole trouble in this case. In the upper part of the back you see a well-marked angular curvature, indicating old caries of the spine. Hip disease and spinal disease not uncommonly exist together, as in this case, but what is still more common and more important is that spinal disease may very closely simulate hip disease. It is quite common for children to be sent up to the Alexandra Hospital with the diagnosis of hip disease, whose hip-joints are perfectly sound, but who are really the subjects of spinal caries. I want to tell you again what I have told you on previous occasions, that you will do well to consider that your examination of any case of supposed hip disease is not complete until you have looked at the spine also. If you follow that rule it will prevent you from making the mistake of treating as hip disease what is, in reality, spinal disease, or from making the error of overlooking the co-existence of spinal caries with hip disease. When I have finished examination of a hip I always turn the child over and look at the back, and that has many times saved me from overlooking such a condition as you see here. Spinal caries may simulate hip disease at several stages. In the early stage the chief complaint may be of pain in the hip and lameness, together with possibly some flexion of the hip. In such a case there may be nothing at all wrong with the hip, the symptoms may be entirely due to caries of the lower lumbar or sacral spine, and there may be little or no obvious deformity of the spine at this stage. I have brought here a museum specimen

showing caries of the upper part of the sacrum, and you will readily understand how caries in that situation may cause no prominent symptoms beyond pain in the hip. Always look with much suspicion on a case of supposed hip disease in which the pain is in the hip only and not in the knee. Of course there are cases of hip disease where the pain is mostly in that joint, but that is uncommon. If the mother of a child with supposed hip disease tells me that the pain is in the hip, I always think immediately of spinal caries, and, upon examination, only too frequently do I find it. Look with some suspicion, too, on any case of supposed hip disease in which the mother is not sure on which side the pain is, or on a case in which she says the pain is sometimes on the left side and sometimes on the right. Such a case as that is probably one of spinal caries. In a later stage, when suppuration is present, spinal caries may also closely simulate disease of the hip-joint. One case which occurs to me is that of a small child whom I saw some years ago on account of supposed hip disease. The hip was stiff, and there was a large abscess round the hip and upper part of the thigh. At first the case looked very like one of hip disease; there was no deformity of the spine, and scarcely any movement could be obtained in the hip-joint on account of the surrounding swelling and suppuration. A slight amount of enarthrodial movement was, however, still present, and caused no pain. I opened the abscess, and when that had healed and the local swelling had subsided, it was found that the hip became perfectly moveable. Eventually a distinct curvature of the spine became visible. It was a case of lumbar spinal caries in which the pus had tracked downwards, and was more obvious in the thigh than anywhere else.

Lastly, hip disease may be mistaken for spinal caries, even in a still later stage when sinuses exist. Probably most of us have heard of a terrible case which occurred in one of the London hospitals some years ago. A patient was admitted with several sinuses about the hip. The surgeon, after what must, I am afraid, have been a very careless examination, amputated at the hip joint, believing the case to be one of hip disease and otherwise incurable. When he had done it, he had the mortification of finding that the hip-joint was healthy. It was a case of spinal caries in which the pus had tracked down to and around the hip.

I could also tell you of a case that I once saw in which excision of the hip had been performed upon the healthy hip of a child who had, unknown to the surgeon, caries of the spine.

The next patient is a boy, also with hip disease. His left leg is somewhat shortened and wasted, and it is fixed, and there has been some suppuration, which has ceased. He has recently developed hæmaturia. Hæmaturia in connection with hip disease is due, as far as I have seen, to one or other of three chief causes. It may be due to stone. I have myself had to do lithotripsy upon a child who was under treatment for hip disease. I may say that the boy before us now has been sounded for stone, but none can be found. Another occasional cause of blood in the urine in these cases is granulation tissue at the inner end of a sinus opening into the bladder. Those are cases in which an abscess about the hip has burst into the bladder. That is a possible case here. Here there has been suppuration, and it has healed externally, but it may have left a sinus internally. I do not think that is the case; there is blood in the urine but no pus. Those cases of hæmaturia in which the blood comes from granulation tissue at the inner end of a sinus are usually those in which pus is abundant in the urine and blood is present only in very small quantities. We must not forget that a child with hip disease has a tuberculous lesion, and tuberculous disease of the genito-urinary tract may co-exist with disease of the hip. This patient has not a sign of tubercle of the bladder, but I strongly suspect he has tuberculous kidneys, though there is no actual proof of this. I do not feel any enlargement of the kidney, and so at present the cause of the hæmaturia must remain doubtful. I shall of course have the urine examined microscopically to see if there are tubercle bacilli or pus in it. Here is another specimen of caries of the spine. The patient from whom this came was a child who died of tuberculous disease of the kidney, but whose primary disease was in the spine.

Fibroid Free in Abdominal Cavity.—Jacobs ('Bull. de la Soc. Belge de Gyn. et d'Obst.,' No. 3) records the removal of a fibroid, weighing over twelve kilogrammes, which was free in the abdominal cavity. It received its blood-supply from the adherent omentum. No signs of a divided pedicle could be found upon uterus or ovaries.—*Amer. Journ. of Obstetrics*, February, 1902.

CASE OF SADDLE NOSE TREATED BY SUBCUTANEOUS INJECTION OF PARAFFIN.

By SCANES SPICER, M.D., B.Sc.Lond.,
Surgeon in Charge of the Throat Department, and Lecturer
on Throat Diseases, St. Mary's Hospital.

THE patient, a laundress æt. 25 (Fig. 1), applied for treatment for nasal suppuration and fœtor, which had lasted from childhood. There was no history

FIG. 1.



indicated in Figs. 2 and 4, the latter drawing of a cast taken two months after injection.

The paraffin is a mixture of hard and soft paraffin, made to melt at 48° C. (105° F.), and is sterilised and kept in sealed bottles. The skin of the nose, etc., is cleansed first with alcohol and then with liq. hydrarg. perchlor. The syringe and needle are cleansed and boiled in the steriliser, which is used as a water-bath to heat the paraffin and syringe (a 15 minim German glass hypodermic one was used, like that for injecting tuberculin).

FIG. 2.



of traumatism or acquired syphilis, or undoubted evidence of congenital taint. She had a well-marked tip-tilted saddle nose, and stunting of the nasal framework. Crescentic wrinkles from eye to eye over the bridge of the nose were well marked, and are shown in Fig. 3, drawing of a cast taken on May 6th, 1901, the day before injection. In addition to ordinary methods of treatment for nasal suppuration I proposed improving the shape of her nose by injecting sterilised paraffin, as first suggested by Gersuny, of Vienna. The result is

In this case ten or twelve syringefuls were injected under the skin, some downwards over the nasal bones, some upwards from the sides of the nose into the depressed gap, and the injected paraffin was moulded by an assistant's fingers, so as to shape the part before setting. The syringe was removed for refilling from its socket in the needle which, when once *in situ*, remained there until it was judged enough vaseline was injected at that spot. The skin was then cleaned, and the points of injection sealed up with collodion. There was no

pain, though the nose looked a little tense and brawny. No paraffin passed into the eyelids, apparently, at the time. There was afterwards no pain or inflammation, but in a few days the upper eyelids became somewhat oedematous. This has varied in amount from day to day ever since, and in the left upper eyelid is a little nodule the size of a large shot. This has been cut down on, but it does not seem possible to squeeze it out.

The result, so far as the appearance of the nose goes, is a very palpable bolstering up of the skin over the bony bridge of the nose, and the formation of a very decent sized organ. As it was done

FIG. 3.



now (March, 1902) ten months ago, and remains in the same state as when first injected, it may be regarded, as far as we can see at present, as permanent. It is certainly a great improvement to her appearance, and the patient states her "mother is proud of her" in her altered condition. She alleges that there has been an improvement in her nasal suppuration and general health. The passage of a nodule of paraffin into the upper eyelid is disappointing, and so is the oedema of the lids. The former is not improbably due to the physiological action of the pyramidalis nasi, which would tend to shift movable bodies upwards and into the orbit.

The latter may be explainable on the theory of a blockage of lymphatic vessels by the paraffin, some of which has got divided up into a molecular condition. It should be remarked, however, that the upper lids were inclined to be puffy before the injection. There is no oedema elsewhere in the body. In any future case still more care should be taken to put pressure on the root of the nose at the time of injection, the paraffin should not be any hotter than absolutely needful to mobilise it, and repeated injections of smaller quantities at lowest possible pressure in all probability may be better than injecting the full amount at once.

FIG. 4.



The method appears to offer many advantages over plastic operations for this class of case.

Gersuny injected cocaine before the vaseline, but this can hardly be necessary, I think, in nasal cases, as the only pain is the prick of the needle. In some of his cases of filling out cavities or formation of artificial protuberances, the effect produced by the vaseline remained unaltered in shape or size for many months, the paraffin apparently becoming encapsuled.

The casts and photographs were shown at the Cheltenham Meeting of the British Medical Association in 1901, and the patient was exhibited at the Annual Meeting of the Laryngological Society of London in January, 1902.

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The Index and Title-page for Vol. XIX will be published with the next issue of THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

SPLENIC ANÆMIA.

Delivered at St. George's Hospital.

By H. D. ROLLESTON, M.D., F.R.C.P.,
Physician to the Hospital.

As there have lately been two cases bearing on splenic anæmia in my wards, this interesting disease will conveniently form the subject of this clinical lecture.

Duration of disease twelve years; hæmatemesis for three years; greatly enlarged spleen; weakness; anæmia of the chlorotic type; diminution in the number of leucocytes; improvement under treatment; splenectomy; death on the second day from gastro-intestinal hæmorrhage.

CASE I.—A young man æt. 23, always delicate, was in the Hospital for Sick Children twelve years ago with much the same condition that he now suffers from. For the last four years he has been worse, and during the past three years he has suffered from hæmatemesis at intervals of five months; he has also had frequent attacks of epis taxis. There is no family or personal history of syphilis. As a child he had measles, scarlet fever, and typhoid, but never malaria. He is pale, easily gets out of breath on exertion, and is not sufficiently strong to earn his living. The lungs are normal. The spleen is greatly enlarged, firm, and smooth, but not tender, so there is no active perisplenitis. There appears to have been some slight diminution in the size of the spleen since a febrile (? influenzal) attack. There is a distinct blowing and continuous murmur, resembling the *bruit de diable*, to be heard over the spleen. It is most distinct in the ninth intercostal space, just outside the scapular line posteriorly, and again in the same space just behind the anterior axillary line. It is best heard when the patient holds his breath in full inspiration. There is no

thrill such as was present in a man with a malarial spleen shown by Dr. Raymond Crawford* before the Clinical Society. This murmur is most probably produced in veins, and either in the vasa brevia, which, with the gastrosplenic omentum, have undergone twisting, or in some of the branches of the splenic vein in the hilum of the organ as the result of a kink. In splenic anæmia the splenic vein is thin walled and greatly dilated,† and would therefore readily become twisted on slight alteration in the position of the spleen, for example when the diaphragm descends. In Crawford's case, where there was both a thrill and a murmur, the cause may have been a narrowing or aneurysm of the splenic artery, or possibly an arterio-venous aneurysm in the hilum of the spleen.

There is no enlargement of the liver, jaundice, or ascites, so there is no reason to think that there is any cirrhosis of the liver. The terminal stage of long-standing cases of splenic anæmia may be complicated by cirrhosis of the liver, jaundice, and ascites, a condition sometimes called Banti's disease, after the Italian physician who described it.

The blood on admission showed what is ordinarily called the chlorotic type of anæmia, viz. a diminution in the red blood-corpuscles and a greater reduction of hæmoglobin—red blood-corpuscles 2,400,000, hæmoglobin 25 per cent. The white blood-corpuscles are considerably diminished, 3500 in a cubic millimetre; after an attack of fever the number slightly increased to 4600, but this is very considerably below the normal count, viz. 8000. Under treatment with iron he greatly improved, and his red blood-corpuscles on March 19th were 5,640,000, hæmoglobin 70 per cent., and white blood-corpuscles 3700. This condition of diminished white blood-corpuscles, or leukopenia, is met with in a certain proportion of the cases regarded as splenic anæmia. The urine was normal. The lymphatic glands in the groin are just palpable, but as he is somewhat thin they may after all be normal in size. There is no sign of tuberculosis, syphilis, or malaria. There was no retinal, gingival, or other hæmorrhages to be found.

Believing that in this case of splenic anæmia splenectomy offered a fair prospect of cure, I consulted my colleague, Mr. Shield, who, fully

recognising the gravity of the operation, expressed himself as ready to perform it. This question was discussed at consultations, and the general view was that splenectomy was the logical and justifiable treatment. The patient was quite willing to run the risk entailed by the operation, and accordingly the spleen was removed by Mr. Shield on March 24th, the operation taking twenty-five minutes. The spleen weighed 37 ounces. Microscopically it showed fibrosis with wide-spread proliferation of the endothelial cells, and disappearance of the leucocytic elements. On the following day the patient had a raised temperature, as patients who have undergone splenectomy often do, and his blood showed an ordinary leucocytosis; the next day he had severe attacks of hæmatemesis, and in spite of transfusion on two occasions died. Shortly before death he passed a good deal of bright blood by the bowel. The case will be published later, but I may say that Mr. Shield considered that the hæmatemesis was probably due to mechanical engorgement of the stomach, brought about by interference with the return of blood from the stomach through the vasa brevia veins, which open into the splenic vein. Unfortunately a post-mortem examination could not be obtained.

The case was one of splenic anæmia, which is a rare disease in reality, though it is often imitated by cases of secondary anæmia associated with splenic enlargement. The following case was at first thought to be one of splenic anæmia, but there is little doubt that it is a case of extensive tuberculosis of the spleen.

Grave anæmia; hæmatemesis; enlarged spleen; hypostatic albuminuria; enlargement of lymphatic glands; reaction to tuberculin.

CASE 2.—A country girl, æt. 16, has always been short of breath and subject to palpitation. There is a slight personal and family history of rheumatism. No other members of her family are affected in the same way that she is; there is nothing to suggest hereditary syphilis. For four years she has been subject to epigastric pain and vomiting about half an hour after food. In the summer of 1901 she suffered more markedly from anæmia, and her "stomach swelled." At this time she had hæmatemesis to the extent of a pint; this has recurred about once a month since. She has not menstruated yet, but her anæmic state is sufficient to account

* R. Crawford, 'Clin. Soc. Trans.,' vol. xxxii, p. 234.

† Harris and Herzog, 'Annals of Surgery,' July, 1901, p. 134.

for this, without having recourse to the view that the hæmatemesis is necessarily vicarious menstruation. The girl is anæmic, fairly well nourished, but sallow and short of breath on movement. The blood examination shows red blood-corpuscles 2,833,000, white blood-corpuscles 7000, hæmoglobin 50 per cent. Though treated with iron, arsenic, and as much fresh air and sun as can be obtained by sitting out of doors, the anæmia has steadily increased, and on March 19th she had under two million red blood-corpuscles and 35 per cent. hæmoglobin. The spleen is smooth, greatly enlarged, extending to the right of the middle line and rather below the umbilicus; it is tender on pressure. There is no evidence of gastric ulcer. The liver is normal, and no signs of disease can be found in the lungs. The heart is somewhat dilated, and there is a pulmonary systolic murmur. The urine presented on admission a curious feature; samples voided when lying in bed contained a considerable quantity of albumin, and were high coloured and lithatic, while the urine collected after the patient had been up some hours was light in colour and free from albumin. This is just the reverse of cyclical or so-called physiological albuminuria, where albumin temporarily appears when the patient assumes the erect position. The albuminuria in this case may be spoken of as hypostatic; probably it depends on the enlarged spleen pressing on the left renal vein and producing chronic venous engorgement while the patient is lying in bed, the pressure being relieved when the erect position is assumed. Falkenheim* in 1884 noticed the same kind of intermittent albuminuria in a man with cirrhosis and an enlarged spleen. He found that the patient's position, whether on his right or on his left side, made a great deal of difference as regards this albuminuria. If the patient lay on the left side there was albuminuria, but if he lay on his face or over on the right side, positions in which the spleen might be thought not to press on the left renal vein, there was no albuminuria. I have seen hypostatic albuminuria in two other cases, one of leukæmia, the other of hepatic cirrhosis. In all three patients the albuminuria at one time or another disappeared from the urine excreted during rest in bed.

Since she was admitted enlargement of the

lymphatic glands in the axilla and groins has developed. This shows that the case is not one of splenic anæmia, for in that disease the lymphatic glands are not affected.

It might be thought that it is a case of lymphadenoma affecting the spleen and the deeper lymphatic glands, and only now becoming superficial. But the fact that there has been a definite reaction after the injection of tuberculin makes it highly probable, if not certain, that the case is one of extensive tuberculosis of the spleen with progressive disease of the same kind in the lymphatic glands. Great enlargement of the spleen due to tuberculosis had been successfully treated by splenectomy by Marriott ('Trans. Path. Soc.,' vol. xlvii, p. 96). In the present case the question of splenectomy was raised at the "consultations," but the girl's condition was thought to be so bad as to render it a very desperate measure. In both these cases I have received much help, especially in the blood-counts, from my house physician, Dr. Lawrence Jones.

THE QUESTION AS TO THE EXISTENCE OF THE MORBID ENTITY OF SPLENIC ANÆMIA.

So many conditions, especially in children, may lead to a clinical combination of anæmia with splenic enlargement, that there is considerable danger that secondary anæmias due to syphilis, tuberculosis, rickets, intestinal disturbance, and other causes may be regarded as splenic anæmia, and that as a result the underlying factor may be neglected. This point of view has recently been ably put forth by our Medical Registrar, Dr. S. V. Pearson.* There is no doubt that many cases of secondary anæmia, the underlying causes of which are not manifest, are labelled as splenic anæmia, but this does not prove that there is not a condition presenting sufficiently definite characters to merit a place as a special disease, any more than the fact that achondroplasia and cretinism have often been described as foetal rickets disproves the existence of foetal rickets. Still if we are to recognise a definite morbid entity of splenic anæmia, its characteristics must be definitely stated.

The characters that go to make up the features of splenic anæmia are:

1. Anæmia of the type usually spoken of as

* Falkenheim, 'Deutsches Archiv. f. klinisches Medicin,' Bd. xxxv, S. 446.

* S. V. Pearson, 'The Medical Chronicle,' Dec., 1901, p. 195.

chlorotic, viz. a diminution in the number of red blood-corpuscles with a diminished corpuscular value in the hæmoglobin.

2. Absence of any leucocytosis. Further, the number of white blood-corpuscles is usually diminished (leukopenia).

3. Considerable splenic enlargement, which cannot be correlated with any other known cause, such as leukæmia, syphilis, tuberculosis, malaria, hepatic cirrhosis.

4. The prolonged nature of the disease.

5. The tendency for gastro-intestinal hæmorrhages from time to time.

6. The termination of the disease, if sufficiently prolonged, in hepatic cirrhosis with ascites (Banti's disease).

NOMENCLATURE.

A number of names have been applied to splenic anæmia. From the general resemblance to myelogenous leukæmia and to lymphadenoma (sometimes termed pseudo-leukæmia), it has been called splenic pseudo-leukæmia. It has also been spoken of as splenic lymphadenoma. It is interesting to note that Gaucher* in 1882 described the morbid lesion of the spleen under the title of "l'épithéliome primitif de la rate," and that ten years later, when Bruhl and Debove† described the same disease as splénomégalie primitive, he recognised the identity of the disease. Osler,‡ who has done much to establish the clinical recognition of the disease, says that the term splenic anæmia was first applied to this condition by Griesinger in 1866, and that in America attention was first drawn to it in 1871 by Dr. H. C. Wood. Banti's disease is the termination of the disease in cirrhosis of the liver with ascites (*vide* p. 405).

MORBID ANATOMY.

The spleen is enlarged in all its dimensions, and greatly increased in weight. The average weight of twelve cases of splenic anæmia which I have collected was 61 oz. A case of Bovaird's,§ in which

* Gaucher, 'Thèse,' Paris, 1882, and 'Semaine médicale,' 1892, p. 311.

† Debove and Bruhl, 'Bull. et Mém. méd., Soc. des Hôp.,' Paris, 1892, p. 596. Bruhl, 'Archiv. General de Med.,' vol. clxvi, p. 673, 1891.

‡ Osler, 'American Journ. Med. Sciences,' Jan., 1900.

§ Bovaird, 'American Journ. Med. Sciences,' vol. cxx, p. 383, Oct., 1900.

the spleen weighed 12½ lbs., a very exceptional record, is not included in these twelve cases.

The surface of the spleen is usually smooth, but may have small tags of fibrous tissue on the surface, while adhesions to adjacent organs may be found. The capsule is usually thickened, and, as in most large spleens, may present localised thickenings or lamellar fibromata. Infarcts may occur and give rise to areas of necrosis and later to depressed scars. On section the organ is tougher and firmer than in health.

MICROSCOPIC APPEARANCES.

On examining a section of the spleen a very peculiar appearance is seen; the normal splenic tissue is largely replaced by fibrous tissue and numbers of large endothelial cells, with two or more nuclei. The fibrous tissue is composed of the thickened trabeculæ and walls of the blood-sinuses, while the large endothelial cells are the outcome of proliferation of the endothelium lining these spaces. The cells are large (16—36 μ) with clear protoplasm, and with their nuclei often near the periphery; among them there are giant-cells. The cells may fill up the venous sinuses so that there is considerable resemblance to an alveolar growth, such as an endothelioma or a carcinoma. Gaucher in 1882 originally described the condition as primary carcinoma of the spleen, and fourteen years later Picou and Ramond* described another case of splenic anæmia as a primary carcinoma of the spleen, which they ingeniously suggested was derived from foetal inclusions of pancreatic cells in the spleen, an occurrence which had previously been described by Peremeschko. The morbid changes have also been regarded as an endothelioma.

The endothelial cells may completely fill up the blood-sinuses, while in other parts the sinuses contain red blood-corpuscles between these large endothelial cells. The appearances are well shown in the figure, which is taken from a case of Dr. W. Collier,† of which I had opportunities of studying and cutting sections in 1895.

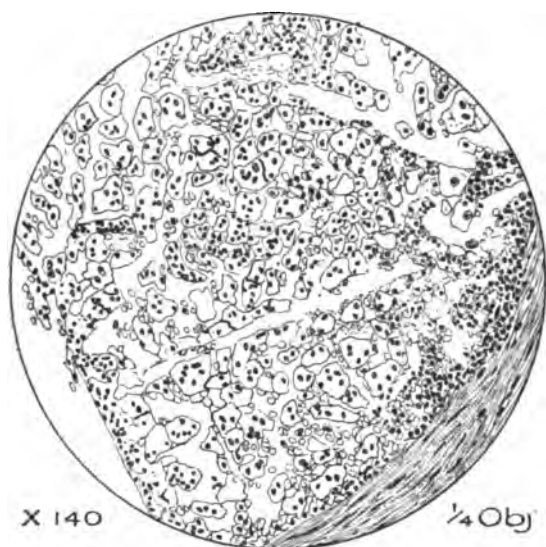
Active karyokinesis has been seen in the large endothelial cells. The large cells sometimes undergo necrosis and no longer take the stain; this is pro-

* Picou et Ramond, 'Archiv. de Méd. expériment. et d'Anat. path.,' 1896, p. 168.

† Collier, 'Path. Soc. Trans.,' vol. xlv, p. 148.

bably due to deficient blood-supply. The proliferation of the endothelium spreads from the sinuses to the vessels which show endarteritis; this process favours thrombosis and cuts off the blood-supply. Endophlebitis of the splenic vein is thus brought about, and by an extension of this process to the portal vein, Banti considered that the terminal cirrhosis of the liver was induced. By an extension of the endothelial proliferation from the splenic blood-sinuses to the lymphatic vessels, a similar change is brought about in the lymphatic glands in the hilum of the spleen.

The Malpighian bodies seem to disappear, and are much fewer than in a normal organ. Probably they undergo the same fibrosis and atrophy that



Blood-sinus filled up with proliferated endothelial cells. The endothelial cells show several nuclei. Between these endothelial cells there are red blood-corpuscles. Near the wall of the sinus there are some lymphocytes.

occurs in chronic infections, and has been produced experimentally by Pilliet.*

The fibrous tissue of the spleen is increased in amount. It has been suggested by Bovaird† that the proliferated endothelial cells eventually form fibrous tissue, and though Harris and Herzog‡ do not support this, the microscopical appearances certainly are compatible with this view. It is perhaps

* Pilliet, 'Comptes rendus Soc. de Biolog.,' Paris, 1894, p. 331.

† Bovaird, 'American Journ. of Med. Sciences,' Oct., 1900.

‡ Harris and Herzog, 'Annals of Surgery,' July, 1901, p. 129.

more probable that fibrous hyperplasia goes on at the same time as the endothelial proliferation, and is due to the same cause, viz. a chronic intoxication. The fibrous tissue of the organ contains pigment granules of hæmosiderin and of hæmatoidin.

The above are the appearances characteristic of splenic anæmia. In some recorded cases fibrosis and atrophy of the pulp and Malpighian bodies have been the only changes described. It is perhaps possible that in such cases the change has progressed, as it may do in lymphadenoma, to fibrosis and disappearance of the endothelial cells. On the other hand, it might be urged that these cases differ essentially in their causation from those showing marked endothelial proliferation. In other words, the condition may have been a secondary anæmia, due to some cause, e.g. syphilis, with associated splenic enlargement. My own feeling is in favour of restricting the term splenic anæmia to those cases found to have endothelial proliferation in the spleen.

The lymphatic glands in the hilum of the spleen and in the neighbourhood of the organ may be firm and enlarged, and may show similar endothelial proliferation to that seen in the spleen.

When the disease has lasted for a long time the liver may show a secondary cirrhosis, due probably to poisons manufactured in the spleen and carried to the liver by the splenic and portal vein. The idea of hepatic cirrhosis being secondary to poisons formed in the spleen has been insisted on by Chauffard,* who, in this connection, regards the portal vein as formed of two branches, the intestinal and the splenic, either of which may convey poisons to the liver capable of inducing cirrhosis.

The liver, though free from cirrhosis, may show the presence of proliferated endothelial cells, free in the branches of the portal vein, and probably conveyed from the spleen. Endothelial proliferation in the lymphatics of the portal spaces has been observed (Bovaird).†

The marrow of the shafts of the long bones may show the same compensatory change, viz. an extension of the red marrow, as in pernicious anæmia. In some cases, however, this has not been found.

* Chauffard, 'Semaine méd.,' May 24th, 1899.

† Bovaird, 'American Journ. Med. Sciences,' Oct., 1900, p. 377.

The condition of the bone marrow in splenic anæmia requires further investigation.

Explanation of the morbid appearances.—The changes seen under the microscope are those of endothelial proliferation, and have lately been compared by Harris and Herzog* to those in diffuse lymphangioma. It resembles to a certain extent the large-celled hyperplasia of lymphatic glands seen in some cases of non-virulent tuberculosis and in other irritative conditions. It also agrees with the histological characters of the soft form of lymphadenoma of lymphatic glands as described by Butlin and Andrewes.† The term lymphadenoma of the spleen is thus justified; it is true that general lymphatic gland enlargement does not occur in splenic anæmia, but, as has been seen, the lymphatic glands in the hilum of the spleen may show the same changes as the spleen. Although, structurally, the changes in the spleen resemble those recently described in lymphadenoma, the disease remains very localised and does not generalise. Possibly this is in some degree due to the liver standing between it and the general circulation. The causes of lymphadenoma and of splenic anæmia are quite unknown, but in both cases there is a chronic process of either a toxic, or more probably infective, nature. It seems probable that the real cause when discovered will be found to be either closely allied or identical in the two diseases.

ÆTIOLOGY.

Sex.—The disease has been said to be commoner in the male than in the female sex, but the difference does not seem to be very marked; in thirty-seven cases that I have collected twenty-one were males and sixteen females.

Age.—In thirty-five cases where the age was obtained the average age was thirty-two years. The collection of cases, however, only contained two in children, and thus probably does not fairly represent the evidence of the disease in early life. Although many, if not the majority, of cases of children with anæmia and splenic enlargement are to be regarded rather as secondary anæmias than as true splenic anæmia, the incidence of the latter disease in children as compared with adults is probably not so low as two to thirty-three.

* Harris and Herzog, 'Annals of Surgery,' July, 1901, p. 130.

† Butlin and Andrewes, 'Brit. Med. Journ.,' 1901, vol. ii, p. 1662.

Heredity does not appear to play much part in the causation of the disease. Cases in two sisters are described by Bovaird, while a sister of Collier's patient probably died from the same disease. Dr. Claud Wilson* has published a remarkable genealogical chart showing that six members of a family in three generations had enlarged spleens. These cases have not hitherto been regarded as examples of splenic anæmia, but from examination of their symptoms it appears quite possible that they are.

Diseases and conditions such as syphilis, malaria, and alcoholism do not appear to have any direct bearing on the development of the disease. But it is reasonable to believe that they or other infections or toxic conditions might reduce the resistance of the spleen, and so render it more susceptible to whatever is the cause of splenic anæmia.

PATHOGENY.

Since removal of the spleen may cure the disease it seems clear that the splenic enlargement is intimately connected with the anæmia, and that there is no general infection or intoxication which stands in a primary relation to these two features of splenic anæmia.

It is equally clear that the disease is not due to splenic inadequacy or to suppression of the functions of the spleen, since not only may splenectomy cure the disease, but removal of the spleen for rupture in previously healthy subjects does not induce splenic anæmia.

It has been suggested that the dissolution of blood-corpuscles takes place in the spleen by means of an enzyme manufactured by the endothelial cells of the spleen, and that in splenic anæmia the enormous increase in the endothelial cells gives rise to greatly increased hæmolysis (Harris and Herzog).† This ingenious theory does not explain the cause of the endothelial hyperplasia, while it may be objected that the blood shows exactly the opposite characters to those met with in pernicious anæmia, which is regarded as due to blood-destruction in the portal area.

It seems more probable that in splenic anæmia a chronic infective or toxic process has its headquarters in the spleen, and that the poisons produced there give rise in the first instance to en-

* 'Trans. Clin. Soc.,' vol. xxiii, p. 162.

† Harris and Herzog, 'Annals of Surgery,' July, 1901, p. 130.

dothelial hyperplasia and fibrosis of that organ, and later so inhibit blood-formation as to produce anæmia of the type usually regarded as chlorotic. The fact that a terminal cirrhosis of the liver may supervene is quite compatible with this theory that there is a chronic infective or toxic process going on in the spleen.

As to the cause of this process we are as much in the dark as we are with regard to the essential cause of lymphadenoma. No micro-organisms have yet been found to account for the splenic changes. There is, however, an analogy between the endothelial proliferation and the large-celled hyperplasia seen in some cases of tuberculous adenitis.

CLINICAL FEATURES.

The onset of symptoms is gradual, and consists in shortness of breath, weakness, and pallor due to anæmia. Splenic enlargement probably always precedes the onset of symptoms, but the patient is often quite unaware of the splenic tumour, though in some instances the spleen is so large that it gives rise to a feeling of weight, discomfort, and abdominal distension before anæmia becomes marked. It is probable that splenic enlargement always precedes the development of anæmia and other symptoms, but it cannot be proved to be so in most instances. The general features of the disease, apart from the blood examination, are those of myelogenous leukæmia. The spleen is greatly enlarged, though not as a rule to the same extent as in myelogenous leukæmia; it is firm, smooth, and usually not tender.

In Sippy's* and my case a bruit, probably of venous origin, was audible; very possibly it is by no means uncommon. The enlargement of the spleen is generally progressive, but occasionally it may appear to diminish while under treatment as in my first case.

There may be attacks of colicky pain in the left hypochondrium which may be associated with gastro-intestinal disturbance. Possibly these attacks of pain may be due to traction exerted on the abdominal sympathetic plexus by the enlarged spleen, or to twisting of its peritoneal attachments.

It is important to bear in mind that the superficial lymphatic glands are not enlarged. The

skin and mucous membranes are anæmic; occasionally there is an icteric tint of the conjunctiva, especially late in the course of the disease when cirrhosis of the liver has developed, but many cases of splenic anæmia never are complicated in this way. The skin may become distinctly pigmented; this melanoderma was noted in six out of Osler's fifteen cases. It has been observed to disappear after successful removal of the spleen. It may possibly be due to arsenic taken medicinally, or to irritation of the abdominal sympathetic by the large spleen. There is dyspnoea and weakness due to anæmia, dilated pupils, and frequently dilatation of the heart with a hæmic murmur as in chlorosis. The temperature is normal, the pulse is soft and slightly quickened.

Hæmorrhages, repeated at intervals, are a characteristic feature of the disease. Nasal and gastro-intestinal hæmorrhages are the forms usually met with. In a few instances hæmaturia, without any cause such as calculus, has been noted and may be described as renal epistaxis (Gull). In a few cases retinal hæmorrhages have been seen. Oozing from the gums may occur, and menorrhagia has been met with.

Gastro-intestinal hæmorrhages are both the most frequently seen and the most important. Severe and copious hæmatemesis, accompanied by melæna, may occur at intervals during quite a number of years. In one of Osler's cases there were recurring attacks during twelve years. In the intervals between the attacks there may be very fair health. The hæmatemesis may be so profuse as to be fatal; this occurred in two out of Osler's fifteen cases.

As to the causation of this hæmatemesis, it is certainly not necessarily due to cirrhosis of the liver, for periodic hæmatemesis is a common feature in splenic anæmia, and may prove fatal before hepatic cirrhosis, which is a late and only an infrequent event in splenic anæmia, has appeared.

It has been thought that the hæmatemesis is mechanical (Osler*) and due to venous engorgement of those areas of the gastric mucous membrane which are drained by the vasa brevia and gastro-epiploica sinistra veins. These veins run into the splenic vein and pass through the gastro-

* Sippy, 'American Journ. Med. Sciences,' vol. cxviii, p. 410, 1899.

* Osler, 'American Journal of Med. Sciences,' Jan., 1900.

splenic omentum. It is possible that the enlarged spleen may, by pulling on the gastro-splenic omentum, gave rise to torsion of these veins and thus obstruct the venous return from the stomach. It is also conceivable that the spleen might, like a wandering spleen, induce kinking of the splenic vein. If this occurred at a point between the entrance of the vasa brevia and the junction of the splenic with the portal veins, intense venous engorgement of the cardiac end of the stomach would result. This might be followed by hæmatemesis.

The possibility that a toxic condition of the blood may play a part in hæmatemesis must, however, be borne in mind on the ground that epistaxis, which also occurs, cannot be explained on purely mechanical grounds.

The source of a fatal hæmatemesis has been found to be an ulcerated varicose vein near the lower end of the œsophagus in a patient whose liver was not cirrhotic.

Ascites may be present and require tapping, while the liver is quite free from cirrhosis. Possibly the ascites may be due to chronic peritonitis depending on irritation produced mechanically by the large spleen.

The urine is nearly always free from albumin. Crystals of uric acid are not deposited from the urine as is so frequently seen in leukæmia, where the excess of uric acid may be correlated with the increased number of white corpuscles and supply of nuclein. In the late stages the urine becomes diminished in amount and high coloured from excess of urobilin.

THE BLOOD.

There is a diminution of the number of the red blood-corpuscles, but it is not excessive and does not approach that seen in pernicious anæmia. The average of Osler's fourteen cases was 3,336,357.

There is a relatively low hæmoglobin worth of the red blood-corpuscles. The anæmia is therefore of the same type as that usually considered to be characteristic of chlorosis, viz. a greater diminution in the amount of the hæmoglobin than in the number of the red blood-corpuscles. Poikilocytosis is occasionally seen.

The absence of any increase in the number of white corpuscles is an important and essential

feature of splenic anæmia. Cases of splenic enlargement with anæmia and leucocytosis should not be termed splenic anæmia, but should be regarded as secondary anæmias. The inclusion of such cases under the heading of splenic anæmia only leads to confusion. In nearly all cases of undoubted splenic anæmia there is not only an absence of any increase in the number of white corpuscles, but an absolute diminution or leukopenia.

In some of the published differential counts the polymorphonuclear leucocytes are diminished, and the mononuclear relatively increased.

It should be mentioned that in his recent work ('Clinical Pathology of the Blood,' p. 231) Ewing states that he has found the blood changes in splenic anæmia indistinguishable from those of lymphadenoma and gumma of the spleen.

DURATION.

One of the essential characters of splenic anæmia is the extremely prolonged course of the disease. It may last for twelve or thirteen years, or even for a longer period. In Gaucher's case the splenic enlargement had been recognised for twenty-five years. This contrasts with the comparatively rapid course of myelogenous leukæmia.

PROGNOSIS.

The disease runs a very prolonged course, and in some instances it is possible that it may cease to advance or become arrested for long periods. In Gaucher's case, where the history went back for twenty-five years, death occurred from pulmonary tuberculosis. If Dr. Wilson's six cases of hereditary enlargement of the spleen belong to this group, they must be regarded as very favourable examples of the disease, for three of them (two alive at the time of his publication) had had the splenic enlargement for more than twenty-five years.

In most of the cases, however, the disease is steadily, though slowly, progressive. Profuse hæmatemesis may prove fatal, while the patients' debilitated and anæmic condition expose them to acute infections which may carry them off before the disease has run its course. If uninterrupted the disease may terminate in cirrhosis of the liver, with ascites or Banti's disease.

DIAGNOSIS.

The *diagnosis* of splenic anæmia has to be made from a number of different conditions, especially from all secondary anæmias associated with enlargement of the spleen.

From *myelogenous leukæmia* an efficient examination of the blood ensures a correct diagnosis. In most exceptional instances the blood-changes characteristic of leukæmia may disappear (Osler); it is just conceivable that a latent case of myelogenous leukæmia might be regarded as one of splenic anæmia. There is no doubt that the most important condition to eliminate in the diagnosis is myelogenous leukæmia, since splenectomy, which has met with some success in the treatment of splenic anæmia, is almost universally and rapidly fatal in leukæmia.

From *pernicious anæmia* the blood examination should distinguish splenic anæmia; further, enlargement of the spleen is not a constant event in pernicious anæmia and is very rarely a prominent clinical feature.

In *lymphadenoma*, or *Hodgkin's disease*, there is enlargement of the lymphatic glands, either local or general, and sometimes moderate enlargement of the spleen. If the deep abdominal glands and the spleen were exclusively affected, the diagnosis would be extremely difficult; possibly such cases do occur, but they are extremely rarely recognised. As a rule some superficial glands are enlarged; this puts splenic anæmia out of court.

In *malaria* the history, the fever, and the detection of the parasite in the blood should establish a correct diagnosis.

Syphilis, especially the tertiary form, in a patient who has not been treated, or only imperfectly so, may produce profound anæmia and very considerable enlargement of the spleen from lardaceous change, from gummatous infiltration, which, by the way, is a rare condition, or from both combined. In a suspected case a thorough anti-syphilitic course of treatment may clear up the diagnosis. That there may be considerable resemblance clinically between the manifestations of syphilis and splenic anæmia is shown by Dr. Coupland's * case, where the spleen of a woman with anæmia was removed with great improvement to her health. The spleen showed fibrosis and atrophy of

the Malpighian bodies. Two years later she died with ascites and hæmatemesis, and was found to have syphilitic disease of the liver.

Tuberculosis of the spleen.—In rare instances, of which Collet and Gallavardin* have collected eight examples, tuberculosis may give rise to very great splenic enlargement. This massive tuberculosis of the spleen might conceivably imitate splenic anæmia, and in a doubtful case tuberculin might be employed to assist in the diagnosis. In massive tuberculosis there may be fever and there may be an absolute increase in the number of red blood-corpuscles, either in association with or independent of cyanosis. This plethora is a very curious phenomenon indeed; in one case, without any cyanosis, there were 8,200,000 reds.

From cirrhosis of the liver with splenic enlargement. In ordinary or portal cirrhosis the spleen is very rarely so much enlarged as in splenic anæmia. It must, however, be remembered that cirrhosis of the liver may supervene on splenic anæmia (Banti's disease). It may be very difficult to tell whether this has occurred, since hæmatemesis and ascites may both occur in splenic anæmia in the absence of cirrhosis. Enlargement of the liver and distended abdominal veins around the umbilicus are in favour of cirrhosis. Osler points out that in cases where hepatic cirrhosis supervenes in hæmochromatosis the spleen may be large and the resemblance to splenic anæmia, which is heightened by the pigmentation of the skin, be considerable. In hæmochromatosis there may be so much concomitant fibrosis of the pancreas that there is glycosuria (diabète bronzé). This is not met with in splenic anæmia. In some rare instances of hypertrophic biliary cirrhosis the spleen is considerably enlarged before the liver becomes affected; such cases have a close resemblance to the sequence of events in Banti's disease.

Gastric ulcer.—The recurrence of hæmatemesis may at first sight suggest chronic gastric ulcer, especially in adult men, in whom the symptoms do not exactly conform to those of the disease as seen in young women. The detection of the enlarged spleen, should, however, lead to a revision of the diagnosis. Some years ago, a man of about forty was under my care with marked anæmia, a tumour in the splenic region, absence of marked gastric tender

* Coupland, 'Brit. Med. Journ.', 1896, vol. i, p. 1445.

* Collet and Gallavardin, 'Archiv. de Méd. expériment et d'Anat. path.', 1901, p. 191.

ness, and repeated hæmatemesis, from which he died. At the autopsy he had a chronic gastric ulcer, and a large hydatid cyst of the left kidney. The association led to an imitation of splenic anæmia sufficiently close to mislead me.

When obstruction or *thrombosis of the splenic vein* occurs as part of thrombosis of the portal vein, the spleen is greatly increased in size, and hæmatemesis is likely to occur. The resemblance of such a case when first seen to splenic anæmia might be misleading, but, as a rule, the course of portal thrombosis is rapid, while that of splenic anæmia is very prolonged. But in exceptional instances a patient with portal thrombosis may live for many years with splenic enlargement and recurrent attacks of hæmatemesis. Langdon Brown* reports the case of a woman who had had hæmatemesis at intervals of ten months for twenty years. The portal and splenic veins were obliterated. Portal thrombosis had been diagnosed by Sir W. Jenner twenty years before.

TREATMENT.

It is difficult to treat a disease the nature of which is not thoroughly known, unless clinical experience has established the empirical fact that a certain drug has a definitely beneficial action. In splenic anæmia there is no drug known that has a specific action on the disease.

The treatment of splenic anæmia at present falls under two heads—(1) the general hygienic or medical treatment, and (2) the surgical removal of the affected organ.

Medical treatment consists in improving the general health by fresh air, sun, good food, and giving drugs to counteract the anæmia, such as iron and arsenic. Improvement may take place after a course of iron, but a permanent cure must not be expected. Some patients bear arsenic badly, and in such cases it should not be persisted in. Inhalations of oxygen have been found to be beneficial.

The surgical treatment, viz. removal of the spleen, is a more heroic but at the same time more logical method of treating a disease in which the morbid process is thought to be one of chronic infection localised in the spleen. The removal of the focus of disease is then analogous to opening

an abscess which is giving rise to septic intoxication. Before splenectomy is resorted to, care must be taken to eliminate leukæmia, for removal of the spleen in that condition is almost always fatal from secondary hæmorrhage.

The results of removal of the spleen in splenic anæmia are encouraging. Thus in nineteen cases collected by M. L. Harris and Herzog* there were fourteen recoveries from the operation, and four deaths, while in one instance the result was not given.

After splenectomy in splenic anæmia the blood condition improves. It has been noticed (Harris and Herzog) that there is an increase in the eosinophile cells as shown by a differential leucocyte count; in one of their cases the eosinophile cells formed 14·4 per cent. as against the normal 2 per cent. There was also an increase in the large mononuclear leucocytes.

After removal of a previously healthy spleen for traumatic rupture in man,† a condition which might otherwise prove fatal from intra-peritoneal hæmorrhage, leucocytosis with increase in the eosinophiles, together with fever, anæmia, enlarged lymphatic glands, debility, and pains in the long bones occur during the period that compensation for removal of the spleen is taking place in the lymphatic glands and possibly in other parts of the body. After splenectomy for splenic anæmia the after-results are much less prominent; indeed, except for the alteration in the differential blood count, some leucocytosis, and temporary fever, there may be little to note. This is probably satisfactorily explained on the hypothesis that owing to the altered condition of the spleen gradual compensation has taken place during the prolonged course of the disease, and has not to be suddenly brought about after splenectomy. The same freedom from after-symptoms is noticed after removal of floating spleen, where, from repeated attacks of acute venous engorgement, depending on twisting of the pedicle which affects the thin-walled vein more than the artery, there is fibrotic atrophy of the tissue of the organ.

That the operation, however, is not one without grave risks is shown by the fatal result in this case where the patient was in a fair state of health at the time, and the operation itself was most successfully carried out.

* M. L. Harris and M. Herzog, 'Annals of Surgery,' July, 1901, p. 133.

† B. Pitts and Ballance, 'Clin. Soc. Trans.,' vol. xxix, p. 102.

* Langdon Brown, 'St. Bartholomew's Hospital Reports,' vol. xxxvii, p. 155.

WITH DR. F. J. SMITH IN THE WARDS OF THE LONDON HOSPITAL.

FEBRUARY 28th, 1902.

A CASE OF BLACK WATER FEVER.

WE have just had in the wards a very interesting case of this disease, which is very rarely seen in England. The patient was a man æt. 29, who had spent some time in Nigeria, on the West Coast of Africa, where he had many attacks of malaria, and probably of actual black water fever. He went there in May, 1900, and had his first attack of malarial fever in July, 1900, and succeeding attacks were pretty nearly at the rate of one each month, but of all these, he says, he has only had two in which he passed black water.* During this time he used to take about five grains of quinine a day as a prophylactic. He was admitted by the request of Dr. Esmond, of Walthamstow, on February 15th, a fortnight ago, and at that time his most prominent feature was the vomiting, which nothing seemed to allay; this symptom, though a common one, is, if persistent, of very unfavourable import. I may very briefly say he remained *in statu quo* until his death, which took place on February 26th. Now a few words to you about the treatment that was adopted. We first of all put him on boric acid, urotropine, and benzoate of ammonia every four hours, but this had no effect. I then obtained some liquid extract of *Cassia Beareana* which had been strongly recommended in an article in the 'British Medical Journal' for February 2nd in this year. All was of no avail, however, as his sickness still continued, so I then tried a dry powder containing one minim of liquor arsenicalis and half a grain of carbolic acid, with some indifferent powder to make it dry. Still there was no reduction in the sickness. Dr. Cross, who has had a large experience in Nigeria of this disease, then kindly saw him and suggested that we should give him plenty of water with bicarbonate of soda in it by the mouth, thus inaugurating a different line of treatment; but still all was of no avail and he sank. The post-mortem showed one or two interesting features: the kidneys more particularly were shaded a deep black or blue in the medulla, with a sort of blue haze over the cortex, evidently malarial pigmentation.

The liver was of a greyish appearance and showed a considerable excess of prussian blue on treatment with ferrocyanide of potassium and hydrochloric acid. Dr. Bulloch has reserved portions of all his organs, and will make a detailed report upon the same. So far as the naked eye was concerned there was nothing beyond this pigmentation to be seen, but Dr. Cross did actually find with Dr. Bulloch some malarial parasites. Those who are interested in the disease will find the little brochure of Dr. Cross* an exceedingly rich mine of information, but for myself I can only say that this is the first case I have seen of it.

I should like to add a few more remarks in defence of the treatment which was pursued, although a fatal result followed. Inasmuch as liquid of any sort seemed to make the patient more distressed and caused repeated vomiting, I knocked off all fluid for forty-eight hours, with a certain amount of success so far as vomiting was concerned, but the patient undoubtedly felt weaker for it. I may say I regret I did not during this time try hypodermic injections of quinine hydrochloride. When Dr. Cross's advice was offered the opposite plan of treatment was pursued, viz. that of placing in the patient's stomach considerable quantities of fluid in the hope that some would be absorbed, but it was too late. The plan would seem to have this to recommend it: that death undoubtedly takes place in some cases from a suppression of urine, independent of the general exhaustion, and Dr. Cross's view was that if the stomach could only manage to absorb a small proportion of the fluid placed in it, independent suppression of urine might be avoided. My reason for not adopting the plan earlier was the distress caused by the vomiting and the fact that he seemed to be suffering from general exhaustion rather than special kidney disturbance.

A CASE OF MULTIPLE PERIPHERAL NEURITIS.

The patient is a girl æt. 17, and her history runs as follows:—She is a servant girl, and she was quite well up to about eighteen weeks before her admission, when she noticed that her feet and legs were beginning to give her a great deal of trouble by aching; this she seemed to think was due to her

* Reprinted from the Epidemiological Society's 'Transactions,' vol. xliii.

work, to standing too much; the aching and tired feeling got worse and worse, and the legs got weaker and weaker, until at last she was unable to walk, and, to use the patient's own expression, her "legs seemed to give way under her and she fell down." She never had any pain except for the continuous aching in her lower limbs, but she felt a certain amount of numbness as if she was walking on things, and she did not seem to mind if people trod on her toes. She was sent up to me at this stage by Dr. Taylor, of Leyton, complaining of the above symptoms and also of a great deal of vomiting. Her condition when I saw her was as follows:—In the legs and arms there was very great loss of power, associated with very marked wasting and a certain amount of anæsthesia in general, the reaction of degeneration was freely made out in those muscles which still gave any reaction at all, both in the arms and legs.

Now let us discuss the diagnosis. The rapidity of the onset clearly, in my opinion, excluded progressive muscular atrophy, which the case at first sight resembled. The case was altogether of too rapid a nature. I then thought that it was possibly a case of subacute anterior poliomyelitis, such as one knows to occur in adult life, but on referring the matter to Dr. Head for diagnosis he decided that it was one of acute infective polyneuritis, and the subsequent progress of the case has, at any rate, not contradicted that diagnosis, for she has now improved very considerably indeed, the process of nerve degeneration having ceased rather abruptly, while regeneration, or at least restitution of function, has rapidly proceeded. Her treatment has consisted simply in rest in bed, with a little general treatment of any unpleasant symptom. The case is one of very great interest from the point of view of diagnosis, which included, to some extent, that of prognosis, for when the paralysis seemed to be creeping up and threatening life, I was told by Dr. Head that if the patient managed to survive the first few weeks after the onset of the disease the prognosis was absolutely good, but that in some cases the progress of the case was so rapid that life was extinguished before the degenerative process had ceased. As regards the exact factor causing the neuritis I can find nothing that would fulfil the purpose. Alcohol was conspicuous by its absence, there was no history of diphtheria, her work did not expose her to any special trade products or

fumes, and one can only look upon it, therefore, as something of an idiopathic nature.

COLITIS WITH BRIGHT'S DISEASE.

This patient is a woman æt. 42, and she was sent to me on account of diarrhœa with heavy albuminuria. On admission the bowels acted five, six, and seven times a day with a large amount of albumin in the urine. I at first took the view that the case was really one of uræmic diarrhœa, and I thought that the prognosis was probably very unfavourable. I at first tried to check the diarrhœa with enemata of hammamelis, but this seemed to quite fail; but after a week or two I tried the effect of an injection *per rectum* of an ounce of bismuth with sufficient starch in the water to hold it in suspension. This seems to have acted very speedily, and has allowed me to modify my opinion of the actual causation of the diarrhœa and, indeed, has led me to believe that we had to deal with a colitis as well as a nephritis. I will now go on to discuss what is the form of nephritis present, and I need not remind you that I have elsewhere* analysed the forms of Bright's disease into those of acute nephritis, consecutive nephritis, the large white kidney, and the cirrhotic kidney. I believe the present case to be one of large white kidney; my reasons for this are—(1) that the albumin was practically always more than half; (2) the amount of urine (and I lay special stress on this point) was always very small, and (3) there was, in addition, the old clinical association, "large white kidney, large white legs." The diagnosis of large white kidney is very important, because I believe it is that form of Bright's disease which has the worst prognosis, and for my own part I believe that it is a disease *sui generis*, and one that invariably ends fatally within from six weeks to as many months.

HEMIPLEGIA.

Here is a case of hemiplegia of rather an important type; it is probably due to syphilitic disease of the vessels of the brain. The patient is a policeman æt. 28, who was admitted under my care with left hemiplegia; it was the second attack, the first having taken place some two years previously. In this patient there is a very distinct

* 'Differential Diagnosis.' By Dr. F. J. Smith. Macmillan and Co.

history of syphilis, and the man has improved very largely under antisyphilitic treatment of mercury and iodide of potassium. But let us consider the treatment of hemiplegia in more general terms. My belief is that 99 per cent. of the cases are due either to hæmorrhage or thrombosis or spasm of vessels, and when we consider our helplessness in dealing with hæmorrhage I think it is but right that the patient should always have the benefit of the doubt as to whether there is specific thickening of the intima of the vessels of the brain or not, and I would treat all of them indiscriminately with iodide of potassium, believing that in this way, if no syphilitic history is obtainable, no harm is done, whereas in those in whom there is a syphilitic history the iodide of potassium often is the only chance. The case now before you was treated in addition with mercury, because I thought that probably he had never been thoroughly, that is for over a year, treated with mercury. There is one point to which I wish now to draw your attention in the diagnosis of syphilitic hemiplegia. I think that in the majority of cases there is more definite information to be gathered from the investigation of the cranial nerves than in any other cause for hemiplegia, inasmuch as they rarely all escape if syphilis be the cause of the trouble. Paralysis of both sixth nerves is not of much use as a factor in the diagnosis, for any intra-cranial pressure is likely to cause this form of paralysis, but if there be paralysis of one or more of the cranial nerves on one side only suspicion of syphilis should be at once aroused. I am well aware that hæmorrhage may thus affect the nerves, or a nerve, on one side; but I believe this is only in an insignificant proportion of cases compared with those in which syphilis is at the bottom of the matter.

INFLUENZA IN ITS REMOTE SEQUELÆ.

A postman has just been sent up to the ward to see me with the history that he had influenza two years previously, and since that time he has always felt in trouble about his work; he is over-anxious, incapable of concentration of thought, frequently forgets to deliver letters, and is very fearful about the correctness of his delivery. Such cases of brain disturbance after influenza are by no means infrequent, and although they present no objective symptoms of disease beyond that on the mental side, I think it is very useful to know what will help

recovery. I have found that a combination of ten grains of bromide of potassium with ten grains of iodide of sodium given three times a day is extremely useful. I believe that the condition is in some way or other due to perverted malnutrition of the neurons, and the iodide seems in some way or other to slowly restore the condition of health. I have treated now a good many cases on the same lines with very good results.

CHRONIC METAL POISONING.

I want now to show you a girl of about twenty-three or twenty-four, whom I have on a previous occasion shown to the Clinical Society as an example of very prolonged and severe anæmia. The patient has been frequently in hospital under my care, and every drug, known and unknown, for restoring the quantity and quality of the blood has been tried in vain. I noticed that during her last visit, some six months ago, to the hospital she had complained to me that her teeth were loose, and I thought at once that it was a case of necrosis with absorption of the alveolar processes. On investigating the matter a little more closely I discovered that she had had for many years (in fact, practically the whole time I have had her under care) some false teeth screwed into the old stumps by means of brass screws, so I at once claimed the aid of the dentist, who removed the loose teeth and the stumps, and fitted her with a plate. The result was extraordinary; she told me she had never felt better for the last ten years than she felt after her last dismissal from the hospital; and although she is still anæmic, there can be no question in my mind that these screws were for many years an unsuspected cause of her ill-health. I might call this case one of warning against cheap dentistry.

WE hear from Dr. Purves Stewart, 7, Harley Street, and Mr. William Turner that it has been decided to inaugurate a post-graduate course of clinical demonstrations at Westminster Hospital during the coming summer session. These demonstrations are fixed for the convenient time of 4.30 p.m. on Tuesday afternoons, commencing on May 13th; they are intended for qualified practitioners only, and the fee for the course of ten meetings is one guinea, but holders of the post-graduate "conjoint" ticket will be admitted without extra payment.

THE LOCAL TREATMENT OF ADENOIDS.*

By J. PORTER PARKINSON, M.D., M.R.C.P.,

Physician to the North-Eastern Hospital for Children.

I HAVE ventured to bring before you to-day a plan of treatment which I have employed for the last three or four years at the North-Eastern Hospital for Children and elsewhere, for a certain class of case usually called adenoids.

The children affected suffer from periodic "colds," generally spoken of by the mother as "bronchitis"; they have attacks of violent coughing, often worse at night or in the early morning, and perhaps followed by vomiting; they often have discharges from the nostrils or ears with slight deafness, occasional headache, and suffer, if of a neurotic temperament, from night terrors.

On examining the chest nothing abnormal may be found, except a slight snoring inspiration and an occasional rhonchus.

The child may have the facial aspect, snoring respiration, and nasal speech of adenoids, and on examining the naso-pharynx with the mirror, or preferably with the finger, the swelling known as adenoids may be felt; in some cases this swelling is hard and fibrous feeling, but in a large proportion it is soft and gelatinous. In the former case there is obviously but one hope for a permanent cure, that is, complete removal of the growth, especially if nasal breathing be much interfered with, or complications such as deafness be present.

In those cases, however, where a soft and gelatinous mass is to be felt, the line of treatment is not so obvious; if left alone, the child in a week or two, with ordinary care, may improve, but the obstruction to nasal respiration will to some extent remain. In these cases which Sir F. Semon, in his recent lectures on "Local Treatment of the Upper Air Passages," alludes to as periodic and transitory, but little has been suggested to hasten the disappearance of the catarrhal symptoms and to relieve the underlying condition. In them, as Sir Felix Semon has pointed out, there may be only a very moderate, or hardly any organised hypertrophy of the lymphoid tissue, but on the slightest provocation so much congestion and engorgement

occurs that for the time all the symptoms of genuine adenoids are closely simulated. In such cases as the above I have been in the habit of applying locally to the naso-pharynx an astringent solution, with the object of producing shrinking of the engorged tissue, so as to allow of nasal respiration being almost at once commenced, and to prevent, if possible, any permanent thickening being left behind owing to the long continuance of a chronic congestion.

I am aware that there is nothing new in this, Mr. Urban Pritchard has suggested the injection of a solution of tannic acid, or a spray containing the same substance, by the anterior nares, but it has always appeared to me that the direct application to the part chiefly affected would be more likely to ensure success, and could have no bad effect on the mucous membrane of the nostrils. Consequently, after satisfying myself by digital examination that the obstruction is of the soft gelatinous variety, I apply a solution containing equal parts of *Liquor ferri perchloridi* and glycerine by means of a brush on a curved handle, the most convenient being the laryngeal brush made by Mayer and Meltzer, which can be bent to such a curve that it can easily be introduced behind the soft palate, and can thoroughly paint the naso-pharynx. With a little practice this can be done rapidly, and with such a minimum of discomfort that the majority of children do not fear its application. This may be done twice weekly at first, and continued once a week till all the symptoms have abated. I supplement this with respiratory exercises such as those suggested by Mr. Arbuthnot Lane, and the administration of a mixture containing cod liver oil and iron, and I believe the symptoms disappear much more rapidly than in cases not locally treated, while the after-history shows that in a large majority of the cases the symptoms have but little tendency to recur, and nasal respiration is permanently established.

In the slighter cases improvement begins after the first application, but many have to be painted more often before the symptoms abate, but when improvement begins it is surprising how permanent it is, for one would naturally expect that the astringent effect would be only temporary; this may perhaps be attributed to the prevention of that permanent thickening which results from long-continued congestion.

* Delivered before the Society for the Study of Diseases of Children.

It is generally admitted that a large proportion of adenoids atrophy spontaneously about the period of puberty, and therefore the nearer the child is to that age, the less permanent is an obstruction likely to be, but it is by no means those cases alone which are benefited by the astringent treatment, for it seems equally efficacious in children under the age of ten years.

In those cases where permanent obstruction is present and the naso-pharynx is blocked by a tough resistant growth, this treatment is naturally of no avail, and removal of the growth is imperative, but in suitable cases the simple method described will be found to give satisfactory results.

Again, in cases which have undergone operative treatment, sometimes more than once, and in which the symptoms have recurred, I have found great benefit ensue.

In conclusion, as may be inferred from what has preceded, I may mention that the term "adenoids" as applied to the above morbid condition is unfortunate, as it implies an organised hypertrophy, which is a very small part of it, and also that the above treatment is by no means intended for those cases of hard adenoids for which removal is the only possible means of cure.

THE new edition of Quain's 'Dictionary of Medicine,' published by Longman's, fully deserves the high opinion formed of its merits. The volume claims to be "a dictionary of medicine, including general pathology, general therapeutics, hygiene, and the diseases of women and children," and the editor, Dr. H. M. Murray, has efficiently secured the fulfilment of that endeavour. Especial praise must be given for the plates illustrating in colours the different bacteria, the morbid appearances of the blood, malarial parasites, ophthalmoscopic appearances, spectra of pigments, urinary deposits, and painful areas in visceral disease. The following extract from Dr. Patmore's article on "Feigned Diseases" is a good example of the excellence of this work:

MALINGERING.

In cases of "malingering," where the feigned disease is the expression of unmitigated imposture, there is always a motive, and the stronger the

motive the deeper-laid will be the plan, and the more determined will be the effort to carry it out, in some cases actual death being risked with a gamester's courage. No station of life is exempt from instances of this form of deceit—if only the motive be strong enough. The most powerful motive is self-preservation; hence we see the most consummately devised schemes of simulated madness presented by persons under trial for murder. Moreover, to evade imprisonment or the more arduous and monotonous duties of military or naval life, the most ingenious devices are often adopted. Fraud upon accident insurance offices is not infrequently attempted in this way. Superannuation from the public service is the motive at times. The escape from responsibility for grave immoral conduct has been the motive for marvellously simulated mental disease. Even young children have at times led practitioners astray by persistently prosecuted physical simulations. Probably a much larger proportion of "epileptic" seizures—as seen among prisoners and mendicants—are instances of malingering than is generally supposed. Paralysis, neuritis, hæmoptysis, hæmatemesis, persistent vomitings, contractions of muscles or of limbs, blindness, deafness, otorrhœa, incontinence and retention of urine—these and an infinite variety of maladies have been feigned. Small stones have been introduced into the urethra, and sand placed in the urine chamber to simulate calculus; persistent pharyngeal ulceration has been caused by applications of nitric acid, as Semon has related; numerous small fragments of boiled chicken bones have been removed from a sinus in the hand; rashes have been produced by cantharides and mustard plasters; the heart's action and the pulse have been excited by violent muscular movements; thermometers have been rubbed in order to simulate a febrile stage; strange and alarming cedemas have been produced and kept up for long periods by ligatures, and extemporised tourniquets have been used in attempts to blanch the limbs, or to waste them. An inexplicable chronic brawny cedema of a limb should always suggest caution in making a diagnosis. As more detailed instances the following may serve:—A well-educated man was sentenced to a long term for a large fraud; on reception in prison he was reported to be acting strangely, and to have drunk his urine and to have

eaten his ordure; he was said to have a most melancholy manner, and to be given to muttering constantly to himself. These symptoms all commenced directly he was convicted. Some marked inconsistencies were observed in his behaviour, and strong suspicions were aroused. The surgeon, upon first visiting him after these "insane" manifestations, gave verbal orders, in the man's hearing, that "as the patient had a preference for a *special* diet, and was *so very insane*, he should be so far humoured as to be allowed as much of that diet as he could himself produce, and any more from other sources he might ask for." Beyond this he was apparently neglected. He did not again exhibit any dirty proclivity, and soon asked for the ordinary prison diet, and offered to work properly.

A robust-looking man suffered from repeated convulsive seizures, accompanied in each case by the expulsion of urine. He was at first treated as a pronounced epileptic, but on being admitted to a new hospital suspicion was aroused as to the genuineness of his case, especially as the man had no marks of past injuries on his limbs. In the hearing of the man the surgeon in charge described in detail to those around the bed certain grotesque and ridiculous phenomena which should follow after a definite interval if pressure were exerted on a special part of the vertex. The appropriate pressure was made, the seconds which followed were gravely counted by the watch, and at the appointed time the man exhibited the precise phenomena that had been described, ending by carrying out his conception of opisthotonus by travelling over the floor of the ward on his head and heels. He was put back to bed, and little notice was taken of him. No medicine and but little food arriving, the fits did not recur, nor did any unusual discharge of urine take place, the man leaving the hospital cured in about five days.

Malformed Children.—M. Schwab holds that malformed children are likely to be born of mothers who have been attacked by variola during pregnancy. He gives a history of a case of congenital hydrocephalus where the mother had passed through a severe attack of smallpox. He also holds that attacks of the other infectious diseases produce, or are likely to produce, malformed children.—*Canadian Practitioner*, March.

EUSTACHIAN CATHETERISATION.

To the Editor of the CLINICAL JOURNAL.

SIR,—To those whose practice includes a large number of patients with diseases of the ear the passage of the Eustachian catheter becomes sometimes necessary.

It has occurred to me that there is one point to which little attention is apparently given, as one never hears it mentioned, nor finds any reference to it in books. To pass the catheter, it has to enter into both nares, passing through the anterior, and, if the operator be not skilful, may remain a little time in the posterior nares before the orifice is found. Therefore, what is likely to happen is this: supposing you have an unhealthy nares the catheter, as it is passed along the floor of these cavities, picks up, or possibly becomes filled at its end, with mucus, pus, or what not, to be finally blown into the Eustachian tube by the air bag, adding to the mucus already in the tube, or putting some there as a start. The possibility of infection must also be considered. The remedy, then, is this: the aurist should douche the nose well with a fluid suited to the case before using the catheter, and, finally, he should have a catheter fitted with a stylet, so that when it is passed it is apparently a solid instrument. When it is in position the stylet is removed and the treatment proceeded with.

I am, Sir, yours faithfully,

MAYNARD GABE, L.S.A.Lond.

Merthyr Tydfil.

THE following passage from the 'Journal' of the American Medical Association of March 22nd contains a remarkable proposal:—"Sharp recommends the performance of vasectomy in hereditary degenerates liable to propagate their defects. He has performed it on forty-two patients, and claims that it does not impair the sexual power, but improves the physical and mental condition. The method he has adopted lately is the English one which selects the scrotal region as the site. Grasping the vas between the thumb and index finger, make a longitudinal incision about three-eighths of an inch long and sever the vas. This ends the operation, and the scrotal wound is not closed. He believes the authority should be given to render every male sterile who passes as an inmate the portals of the almshouse, insane asylum, institute for feeble-minded, reformatory, or prison.

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